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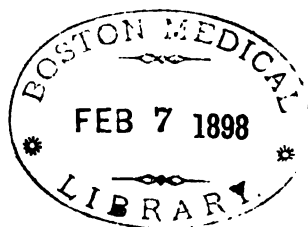
EDITED BY

L. ELIOT CREASY, M.R.C.S.Eng., L.R.C.P.Lond.

20, HANOVER SQUARE, LONDON, W.

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THE CLINICAL JOURNAL.

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ON SOME PAINFUL AFFECTIONS OF THE FEET.

By A. H. TUBBY, M.S.Lond., F.R.C.S.Eng.,

Assistant Surgeon to, and in charge of, the Orthopædic Department, Westminster Hospital; Surgeon to the National Orthopædic Hospital; Surgeon to Out-patients, Evelina Hospital for Sick Children.

PART I.

IN this series of articles it is my intention to describe, and give the treatment of some painful affections of the feet, of fairly common occurrence, which arise not so much from injury as from alterations in the relationships of the bony surfaces, that is to say, affections in which the pain is due to pressure falling upon parts which are not adapted for it. I shall exclude affections arising from diseases of the bones and joints and primary inflammatory affections of the skin. One of the first signs of undue pressure is the formation of corns. Taking, for example, corns on the anterior part of the sole of the foot; if, for instance, a row of corns be found beneath the heads of the metatarsal bones, either right-angled contraction of the tendo Achillis or contraction of the plantar fascia causing talipes arcuatus is present. If one or two corns are found under the heads of the second, third, or fourth metatarsal, and not on the first and fifth, then metatarsalgia, or Morton's disease, should be suspected. Right-angled contraction of the tendo Achillis is the first degree of talipes equinus. In the first place, I would briefly describe this latter affection. So far as the production of pain and lameness are concerned, it is not proposed to fully describe in all its aspects talipes equinus, except in so far as it interferes with locomotion and causes considerable pain.

Talipes Equinus.

The foot should normally be capable of dorsal flexion on the leg to the extent of eighteen degrees beyond the right angle; any condition in

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which this angle is diminished at the ankle-joint without inversion or eversion is correctly called talipes equinus. A few degrees of diminution of dorsi-flexion are not of importance, but if the foot cannot be flexed beyond the right angle then there exists,

The First Degree of Talipes Equinus, or right-angled contraction of the tendo Achillis. In the examination of these cases it is essential to note that in estimating the angle of flexion of

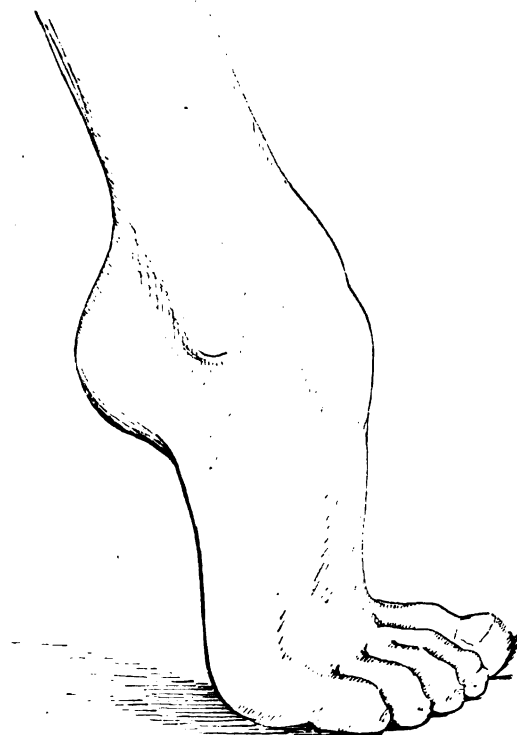


Fig. 1.—Paralytic Talipes Equinus.

the foot the knee must be kept fully extended, since shortening of the tendo Achillis is readily compensated by flexion of the knee. Unless the whole foot can be brought squarely to the ground with the heel in complete apposition without pain or force, and with the knee fully extended, the first degree of talipes equinus exists. The results of this slight deformity are (1) the formation of corns beneath the heads of the metatarsal

bones'; (2) slight lameness and shortening of the stride. For ease in walking, complete flexion at the ankle is necessary; and if the knee is kept slightly flexed the stride is necessarily lessened.

(3) As the corns increase in size, disability in walking, and considerable pain are felt, both about the heads of the metatarsal bones and also in the arch of the foot.

The Second Degree of Talipes Equinus.—(Fig. 1) The heel is raised well off the ground, and a transverse crease is seen above it. Progression takes place on the heads of the metatarsal bones, and decided lameness is present, due partly to the

degree in the spastic; the head of the astragalus forms a distinct prominence on the dorsum of the foot.

The Third Degree is an exaggerated condition of the second; so much extension is present that locomotion takes place on the dorsum of the foot, and it is bent completely backwards, and the toes are, as it were, "tucked underneath." In this stage very considerable pain indeed is present, owing to the formation of the corns and to the presence of numerous false bursæ. In some cases ulcers are found at the sites of pressure. The varieties of talipes equinus are, firstly, spastic, due

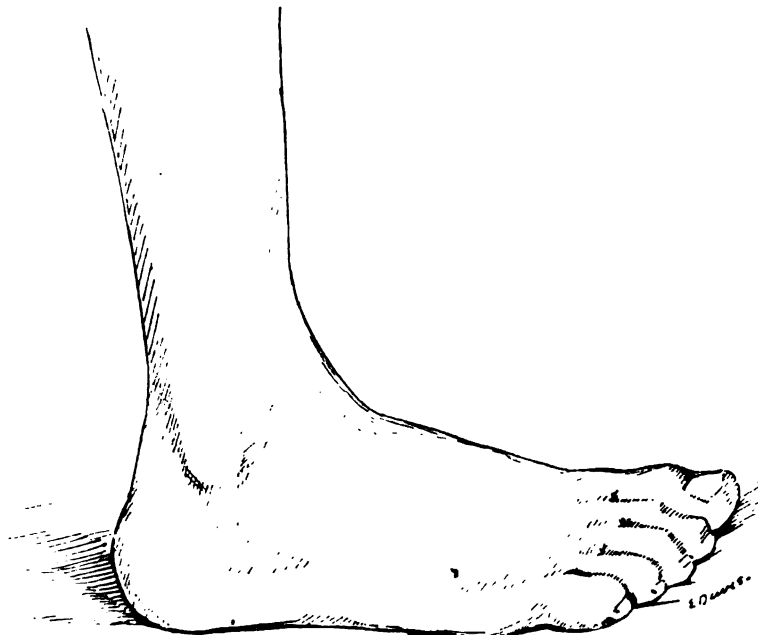


Fig. 2.—The results of treating the foot in Fig. 1 by section of the plantar fascia and tendo Achillis.

extended foot, and partly to large and inflamed corns. In congenital and spastic cases a broadening of the front part of the foot, owing to the spreading of the heads of the metatarsal bones and separation of the toes, may be seen.

The heel is ill-developed, and the skin is quite thin and shows no signs of pressure; the tuberosities of the os calcis are absent, and so, too, is the natural pad of fat in that situation. The limping gait in paralytic cases, and the jerky step in spastic cases, are characteristic. The plantar fascia is frequently contracted in this stage, notably in paralytic cases and to a less

to hemiplegia and spastic paralysis; secondly, paralytic, from infantile paralysis; and thirdly, the congenital cases. The last-named are, however, very rare indeed; in fact, congenital talipes equinus is the rarest of all forms of talipes. There are other rare causes, for instance traumatic, due to injuries of the ankle-joint; cicatricial, from burns on the back of the leg; talipes decubitus due to retention of the foot in a vicious position, as for instance the pointed feet of a bedridden patient.

The Treatment of Talipes Equinus.—In cases of the first degree, or right-angled contraction, mani-

pulation and exercises may be employed, but time and trouble are saved by division of the tendo Achillis. If there is any contraction of the plantar fascia this should have been previously divided.

In equinus of the second degree, division of the plantar fascia, followed by tenotomy of the tendo Achillis, is called for (Fig. 2). It is advisable to make sure that the front part of the foot and the heel can be placed in one plane before the tendo Achillis is divided. The condition of the toes varies. Sometimes they are in a straight line with the metatarsal bones, at other times they are clawed. If this condition of clawing is present, and the extensor muscles show by electrical tests that there are but slight signs of degeneration,

part of the foot must be unfolded first, then the back part of the foot is let down, so to speak, by the section of the tendo Achillis; but before tenotomy is entered upon, time must be allowed for the inflammation of the skin round the false bursæ to subside, and then the treatment directed to the restitution of the deformed foot may be properly commenced.

The treatment of spastic and congenital cases is conducted on the same lines.

Talipes Calcaneus.

This deformity is the opposite of talipes equinus, but inasmuch as the heel is that part which is naturally designed to bear the pressure of the body, but

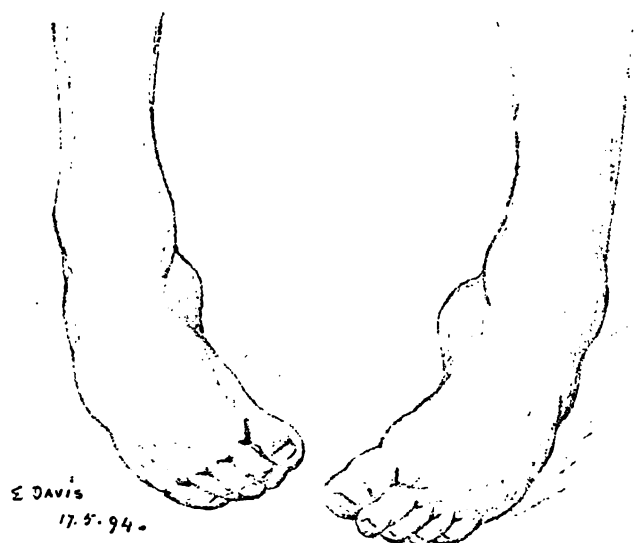


Fig. 3.—Talipes Equino-varus.

the extensor tendons should be divided at the roots of the toes at the same time as the plantar fascia is severed. The immediate after-treatment consists in the use of plaster of Paris or of Scarpa's shoe. As soon as the union of the tendo Achillis is quite firm, the patients may be allowed to walk. But it is well to point out that in many of these cases, especially paralytic ones, the tendo Achillis is apt to elongate again after division, if the patient be allowed to walk about too soon; and it is well in that case to order a walking-instrument, with a half-stop at the ankle to prevent undue dorsal flexion at that joint. This should be worn for at least a year after the operation.

The Treatment of the Third Degree.—The front

little pain and disability result. I have, however, seen corns over adventitious bursæ on the heels and over the tendo Achillis in severe cases, as the patient tends to walk, not on the heel, but on that part just above it. In paralytic cases the integument is also cold, blue, and liable to chilblains. But little can be done by way of treatment beyond an elastic arrangement to raise the heel.

In most cases of so-called talipes varus the tendo Achillis is contracted; hence these cases are more justly named equino-varus, and we may now consider these in so far as they may be included under the term painful affections of the feet.

Talipes Equino-varus.

The causes of this deformity are either congenital or acquired. In the latter form there is a further subdivision into spastic, paralytic, traumatic, and articular.

Paralytic equino-varus is due to acute anterior poliomyelitis, and traumatic equino-varus is the result of fractures, dislocations or separations of the epiphyses at the lower end of the tibia and fibula. The common varieties of talipes equino-varus are congenital, spastic, and paralytic. The most inveterate cases of equino-varus which come before our notice are of the congenital variety.

when the mother observes early that the child's feet are turned somewhat inward, and seeks advice. But if the child has once been allowed to walk for any length of time, then the feet become painful on the outer borders, and considerable trouble results. In one inveterate case of talipes equino-varus of a congenital nature, which came before my notice, the skin of the external border and dorsum of the foot was thickened and studded with corns, which were inflamed, and which suppurated from time to time and rendered locomotion almost impossible. Between the skin and the bones, bursæ had formed which were also



Fig. 4.—The case in Fig. 3 after treatment.

Now the deformity consists of the following departures from the normal: the heel is raised and the foot is extended,—that is, it is in the position of plantar flexion,—the sole of the foot and the toes are adducted and brought to the middle line, instead of being directed to the front; the internal border of the foot is raised to a varying degree and bent upon itself, so that it is concave; the external border of the foot is convex, and forms the main point of support in progression (Fig. 3). It is largely owing to pain arising from the existence of corns and false bursæ on the outer border of the foot that these cases come under notice, except

inflamed, and it is on record that the suppuration has extended to the joints of the foot and has caused extensive disorganisation of those parts, resulting finally in amputation.

As a matter of convenience we may divide talipes equino-varus into four degrees.

Of the *first* degree are the slight cases in which the foot can be momentarily replaced by manual force, and it is necessary to remark that infants who have not commenced to walk usually keep the foot in a position of slight inversion.

In the *second* degree the foot cannot be replaced manually, and on attempting forcible

reposition there remains some adduction or extension of the foot, and the sole cannot be planted squarely on the ground, while the great toe is much separated from the second.

In the *third* degree, which is seen in children and adults, the foot is in a rigid and resistant state, with strong contraction of the soft parts.

In the *fourth* degree the deformity is inveterate and of old standing, and much malposition of the bones is present.

Division into these degrees is valuable from the point of view of treatment.

Treatment of the First Degree of Congenital Equino-varus.—Such cases can be treated by manipulation alone, or by manipulation combined with massage and retentive apparatus. The movements to be practised in manipulation are abduction and eversion at the transverse tarsal and sub-astragaloid joint, with flexion and extension of the whole foot at the ankle, finishing up with circumduction. In all these movements care should be taken that the grasp of the left or fixing hand of the surgeon is made by the thenar eminence and the whole length of the opposing fingers, and not by the tips alone. Treatment by manipulation alone calls for a considerable



Fig. 5.—Malleable iron splint.

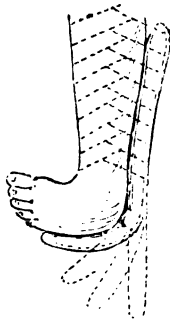


Fig. 6.—Showing the method of everting the foot by the malleable iron splint.

amount of intelligence and persistence. As an adjunct, manipulation is also valuable in the after-treatment of the more severe degrees. Manipulation may be combined with retentive apparatus. It is largely my custom to employ the flexible metal splint (Figs. 5 and 6). This is a straight well-padded piece of soft iron, strong enough to neutralise the contracted muscle, but sufficiently flexible to be bent to any angle. The method of application is as follows:—The splint is first

bent so as nearly to fit the outer border of the leg and foot. It is then, by three or four turns of a bandage, fixed to the leg, and the foot is slowly abducted to the splint, and held in position by other turns of the bandage. Gradually the angle of the splint can be diminished until the foot can be fixed in a straight line with the leg, without pain. This suffices to control the varus.

For the equinus the splint should be bent to the right angle, and fitted to the back of the leg and sole of the foot. In this stage it is better to employ two splints, one in the manner just mentioned, and the second one on the outer side of the leg and foot. The splints are to be worn day and night, being removed for a short time twice daily, when manipulation is practised, and immediately re-applied.

The duration of this form of treatment is about two to three months in slight cases, but considerable care and the use of retentive apparatus,—such, for instance, as a rectangular tin-shoe,—are necessary if relapse is to be prevented. The case should be carefully watched for some years in order to prevent relapse during the period of growth.

The Treatment of the Second Degree.—This is effected by tenotomy, either with or without wrenching the foot by the hand, and the after-use of retentive apparatus. It is most commonly advocated that the treatment of congenital equino-varus should be conducted in two stages: firstly, complete reduction of the varus portion, leaving the tendo Achillis untouched; secondly, when the varus is overcome, division of the tendo Achillis to reduce the equinus. The object of thus dividing the treatment into two stages, and leaving the tendo Achillis till last, is to use the back part of the foot as a fixed point from which to unfold the front part. In some cases of this degree the plantar fascia will be found contracted, and section of it is necessary; so that the line of treatment which I find most advisable in these cases is as follows:—Divide the plantar fascia if it is contracted, and at the same time divide the tibialis anticus and posticus tendons near the ankle. The foot may then be placed in one of the malleable iron splints which I have mentioned above, and extended gradually; or it may be more rapidly extended by the surgeon's hand, and placed for two or three days in plaster of Paris. Then

the plaster-of-Paris bandage is opened, and the foot is manipulated for some ten to twelve minutes, and the corrected position thus obtained is secured by the re-application of the plaster bandage, and so on every three days. When the foot can be brought into a straight line with the leg, then the tendo Achillis is divided, and the heel is brought down, and the leg and the whole of the foot may be placed in plaster of Paris for six weeks or more until the tendo Achillis is firmly united. After that the patient is advised to wear an appropriate walking apparatus, consisting of double uprights from the ground to the calf, a varus T-strap and a half stop at the ankle. A rectangular tin shoe should be worn at night. This point must be attended to, inasmuch as the strong muscles tend to overcome the weak during sleep.

In the *Treatment of the Third Degree* the same tendons are divided; but it is often necessary to resort to the use of the wrench, and I prefer that form of wrench which was invented by Mr. Thomas of Liverpool. By its use the deformity can be quickly and rapidly reduced, and if necessary it may be used on two or three occasions. The same care is required in the after treatment. As the foot is gradually restored to position the advantages gained must be secured by the use of proper retentive apparatus, and some surgeons prefer to use the malleable iron splint I have mentioned, others Scarpa's shoe, or more commonly plaster of Paris is applied.

The Treatment of the Fourth Degree.—Operations on the bones are frequently called for, and the

the outer part of the foot, inasmuch as astragalectomy causes the excessive plantar flexion and excessive inversion of the foot to disappear at once. In only one instance have I been obliged, in addition to removing the astragalus, to take a wedge from the outer side of the foot, and this was a case of relapsed varus which had been operated upon previously. It is very rare indeed for cases of congenital talipes equino-varus to require amputation, and there is no doubt that even the most severe cases can be reduced by time and patience.

Talipes Arcuatus and Plantaris or Pes Cavus.

These deformities increase the concavity of the arch and corresponding dorsal convexity, with the result that the treading surface is considerably



Fig. 7.—Talipes Arcuatus.

diminished, and the body-weight falls upon the heads of the metatarsal bones and the heel (Fig. 7). By talipes arcuatus (Fig. 8) is meant



Fig. 8.—Effect of section of plantar fascia on case in Fig. 7.

operation which is preferable to all others is astragalectomy. It is much superior to tarsectomy, meaning by that term the removal of a wedge from

those cases of pes cavus in which the arch is increased, and when the patient raises his foot from the ground the toes and the heel are on

the same level. In talipes plantaris (Fig. 9) the balls of the toes drop somewhat below the heel.

Many cases of pes cavus are due, in children, to rheumatism—a rheumatic contraction of the plantar fascia. In other instances, however, the cause is slight paralysis of the anterior muscles of



Fig. 9.—Talipes plantaris.

the leg which dorsi-flex the foot; in others, according to Duchenne, the deformity arises from paralysis of the interossei and the lumbricales muscles. But from whatever cause it comes, the ultimate result is contraction of the plantar fascia and increase of the arch. Now when the patient bears his weight upon a foot so affected (Fig. 10), the effect is considerable tension of the



Fig. 10.—The "tread" in talipes arcuatus.

plantar fascia and of the short muscles of the sole of the foot, with the result that with each step there is considerable pain. Talipes arcuatus develops so slowly that it very rarely gives rise to pain and trouble till adolescence is reached. The patient then notices pain in the sole of the foot and up the front of the leg, both of which symptoms are due to the formation of corns beneath

the heads of the metatarsal bones and tension on the plantar fascia, and tendons in the sole of the foot. The pain caused by arcuatus is not unfrequently ascribed to rheumatism, but the corns on the front part of the foot, the firm bands of



Fig. 11.—Painful spasmodic condition of a foot affected with talipes arcuatus.

fascia, and the loss of treading surface ought to indicate the real source of trouble.

The treatment consists in division of the plantar fascia, after which extension should not be made for three or four weeks, as cicatricial thickening and pain are thereby avoided. The best apparatus for after-use is a tin shoe with a divided sole plate, so arranged that the front part of the foot may be uplifted and brought into the same horizontal plane as the heel. The raising of the front part must be done gradually, as some pain incidental to the rapid method is thereby avoided. If there remain much rigidity of the tissues afterwards, a second operation is advisable; or considerable relaxation of the fascia may be obtained by soaking the foot in hot water containing bicarbonate of soda, afterwards manipulating it freely and stretching the sole. The relief afforded by this simple operation of section of the plantar fascia is very considerable indeed.

(To be continued.)

Acne.—Spray with a one-half to one-fifth per cent. solution of resorcin, and follow by the application of an ichthyol plaster; after the disappearance of the acne an ointment of chrysarobin, at first 20 per cent., then 10 per cent., should be applied.—*Pediatrics.*

A NOTE FROM THE CLINIC

OF

MR. PAGET AT THE WEST LONDON
HOSPITAL, FEBRUARY 27th, 1897.

1. Lateral Frontal Meningocele.

GENTLEMEN, — The first case that I have to show is certainly a very rare one. The patient is a boy 8 years old, with a meningocele, which presented itself when he was an infant, not in the middle line, but at the inner angle of the right orbit, coming through a small round opening in the region of the lachrymal bone. The opening was about half an inch in diameter, and the skin was raised over it as a soft rounded swelling, which pulsated, and rose and fell as the child coughed or cried. The case is of importance, as an example of yet another form of swelling that may exist at the upper inner part of the orbit. I have seen a case of nævus in this situation, and a case of dermoid cyst; but here is a third form of new growth in this region, viz. lateral meningocele. The progress of the swelling in this boy's case is worth noting. The gap in the bones has hardly altered since I first used to see him, when he was only nine months old; nor is there more swelling over it than there was at that time. But the breadth between the orbits, and the flattening and widening of the nasal bones, makes a most unsightly deformity. What is worse, he is subject to headaches, and two years ago he had some fits; but these have not recurred. I fear the meningocele will slowly invade yet further the nasal fossa; as in a case that has been recorded, where the disease was actually mistaken for nasal polypus, and so treated, with a fatal result.

Some of these meningoceles may in time cease to enlarge, and may even come to be cured by the growing together of the cranial bones. I showed a case of this kind at the Clinical Society in 1893, but in the present case I am afraid there is no hope of any cure either from nature or from surgical treatment.

2. Ulceration of Nævi.

The next case, a baby four months old, is worth noting as an example of the ulceration of nævi.

The whole upper part of the right ear is disfigured by a raised capillary nævus, which is now somewhat deeply ulcerated over the greater part of its extent. This natural cure of capillary nævi by ulceration seldom does any harm, and in the end it gives results about as good as those obtained by the use of nitric acid. But I have seen one or two cases where the nævus was of considerable size, and the ulceration was in proportion severe and prolonged. And I remember another case, of a new-born infant with a capillary nævus, a "port-wine stain" covering nearly the whole of the right side of the face; it began to ulcerate when the infant was only three days old, and the ulceration invaded the orbit and destroyed the eye, and death occurred within a fortnight of birth. I believe that this ulceration may occur of itself; at all events, one seldom gets any other account given of it, nor does it happen, as has been said, only in syphilitic children. Ulceration of those nævi that we call mixed, having cavernous tissue beneath a capillary surface, may cause them to bleed; but this bleeding is not likely to be severe. Mixed nævi, and venous nævi, unless they are adapted for excision, are best treated by electrolysis; and this electrolysis should be done very patiently, a little at a time, with a long interval between each use of the needles, to get the full effect of the shrinking of the growth; it is impossible to hurry the process, and in the end one gets the best results by working slowly. Capillary nævi, if they ulcerate, are generally to be left to themselves; this natural cure of them will probably give as good a scar as we should get by the use of caustics.

3. Ligature of Gluteal Artery for Secondary Hæmorrhage.

The next case is of great interest. The patient, a boy 15 years old, received a bullet wound in the left gluteal region, with fracture of the sacrum, and laceration of the gluteal artery. As the result of this injury he had incontinence of fæces, and afterwards secondary hæmorrhage, for which the artery was tied; he had also pneumonia, probably from septic absorption, and his general condition, a month after admission, is still very serious. He was brought to the hospital on January 24th, immediately after the accident; the bullet, a large conical one, had entered just below the crest of the right ilium, near the posterior superior spine, and

had passed downward and inward to the sacrum. It was found and removed by Mr. Poynder, the house surgeon, after enlarging the wound; and we hoped that all would go well. For some days he had complete incontinence of *fæces*, but this passed away. Then he had slight secondary hæmorrhage; this was stopped by plugging the wound, but four or five days later it occurred again. I now cut down through the gluteal muscles, and found the gluteal artery cut across and bleeding, just where it issues from the great sciatic notch. I also found the edge of the sacrum broken, and removed some splinters of bone from it. It was easy to lay hold of the bleeding vessel with the forceps, but then came the difficulty of getting a ligature round it, at the bottom of such a deep wound; and for this it was necessary to gouge away the edge of the notch with the cutting forceps. He has had no more bleeding, but a few days after admission he showed signs of pneumonia, which began at the base of the left lung; and a week later there were signs of a patch of consolidation near the apex. Moreover, there is ulceration of the skin over the middle of the sacrum; this is, I believe, not a pressure-sore, but a true destruction of the tissues from injury of trophic nerve-fibres. Altogether, his general condition is very serious. There has been a very profuse discharge from the wound, bringing with it some shreds of clothing and small sloughs; and there is no doubt that he is suffering from septic absorption.

4. Operation for Ununited Fracture of Femur.

The next case is that of a man aged 48, on whom I operated for ununited fracture of the upper part of the right femur. Two things are to be noted about him: first, that he fractured his femur merely by falling over some obstacle, about a foot high, that was on the floor; next, that on his admission, and for many weeks after it, he was feeble, depressed, and wretchedly anxious about himself. He worked at a very unhealthy trade, exposed to the fumes of charcoal; he was ill-nourished, anæmic,—altogether a bad patient. The fracture was of the upper third of the femur, and after many weeks of treatment there was not the very least attempt at union of the fragments. The upper fragment was drawn outward, the lower fragment was drawn upward

and inward, lying high up beneath the vessels; and it was impossible to bring the fragments into anything like good position. I therefore exposed the fracture by very free incision, and found it even worse than we had thought it, for there were three fragments, a long thin shell of bone having been broken off the inner aspect of the shaft, and lying to the inner side of the lower fragment. There was not the least sign of any attempt at union; the fragments were as smooth and devoid of callus as if the bone had only just been broken. I lashed the third fragment to the lower fragment by passing a loop of very thick silver wire round them, and wired the upper and the lower fragments together by drilling them. Happily this succeeded, and he has now an abundance of callus, and firm union.

5. Acute Peritonitis after Operation for Strangulated Hernia.

The next case has given us a great deal of anxiety, but I am glad to say that the patient is well at last, and is going out in a few days. She is 67 years old, and was admitted with strangulated femoral hernia of the right side. The strangulation had lasted from Sunday to Saturday, nearly a week; but her medical attendant had overlooked the hernia, and had treated the case as one of internal obstruction. On admission her general condition was very unfavorable, and in the right femoral region was a tense swelling about the size of a walnut. Operation showed a small knuckle of intestine, very badly damaged, moored by soft bands of lymph to the bottom of the sac: and the sac was inflamed, cedematous, and blotched with hæmorrhages. Though the intestine was very seriously injured, I was justified in returning it, after carefully loosing it from the sac. I did this, and tied the sac and cut it off. That same night the bowels acted freely several times, the vomiting stopped, and we hoped she would go on steadily to recovery. But on the third day after the operation she suddenly became dangerously ill, with vomiting, severe abdominal pain, high fever, and prostration; and we found in the right inguinal and hypogastric regions a large, firm, ill-defined swelling, acutely tender. Her temperature rose next day to 104°, and for many days it seemed hardly possible that she would recover. She was delirious, feeble, passing her motions under her,

and requiring the constant use of the catheter. Then, slowly, she began to mend, the indurated mass in the abdominal cavity gradually disappeared, and she is now at last convalescent, and will leave the hospital this week.

She recovered because she was nursed with the utmost skill and care. The treatment of cases of strangulated hernia after the operation is almost as important as the operation itself. This patient, almost immediately after the operation, was given something by the mouth every hour; only a few teaspoonfuls of stimulant or of fluid food, but still something. These patients suffering from what we may call the asthenic form of strangulated hernia must not be starved after the operation. They are advanced in age, feeble, pulled down by hard work, drink, or chronic organic disease, ill-nourished, and now still further exhausted by pain, vomiting, loss of sleep, and fear of death. Often they come to hospital in the middle of the night, and at once they are subjected to the further shock of the anæsthetic and the operation. Such patients must not be starved, or fed only by the rectum; it is absolutely necessary that we should begin, even a few hours after the operation, to give them small quantities of stimulant and of fluid food by the mouth.

The following cases were also shown:—1. Erosion of knee-joint for tubercular disease; 2. Cancer of tongue and of cervical glands; 3. General tubercular caries with amyloid disease; 4. Superficial gangrene of the feet, from exposure to cold and damp; 5. Inguinal colotomy for cancer of the rectum.

DEMONSTRATION OF CASES

At the Monthly Meeting of the North-West London Clinical Society, held at the North-West London Hospital, March 17th, 1897;

Dr. CUBITT LUCY in the Chair.

Primary Lateral Sclerosis.

Dr. HARRY CAMPBELL showed a woman *æt.* 52, the subject of primary lateral sclerosis. She was married, and had had fifteen children. She had always been strong, active, and healthy, but had

suffered from rheumatic fever twenty-one years ago. Eight months ago she felt a cold sensation in the middle finger of the right hand, and a sensation of cold and weakness spread up the right arm. Two months afterwards the right leg became similarly affected. Subsequently the left arm and leg became implicated. During the last six months she had been unable to walk, and the arms had been growing weaker, the right arm being now almost completely paralysed. She had now marked rigidity in both legs, some rigidity in the upper extremities, increased knee-jerks, ankle-clonus, and exaggerated wrist-jerk and elbow-jerk. A jaw-jerk was also obtainable on the right side. This side of the face appeared weaker than the left. Some of the smaller muscles of the hands, notably the interossei, were atrophied, the hands being somewhat "claw-shaped." These muscles responded feebly to faradism. There were absolutely no sensory troubles, nor bladder nor rectal disorders. His view was that it was a case of primary lateral sclerosis, with right implication of the anterior horns in the cervical region. This was a very rare disease, some observers doubting whether it was ever primary, and he believed he was right in saying that no case had ever been satisfactorily demonstrated post-mortem.

Dr. GUTHRIE said the case was probably one of lateral sclerosis, but he was somewhat in doubt as to whether it could truly be called a primary condition, or really secondary. The disease might be produced by lesions of the lateral columns, but they might also be caused by lesions of the cerebral hemispheres, as illustrated in cases of congenital spastic paralysis, which were usually due to some sclerosis of the motor tracts dependent upon morbid conditions of the cerebral cortex. Considering the condition of the present patient—her thick speech and emotional temperament,—he was inclined to think the condition was primarily cerebral rather than spinal. His first thought was that she appeared like a myxœdematous patient. He would, therefore, like to hear from Dr. Campbell whether he had discovered any further symptoms which might point to that disease. Otherwise he was inclined to regard the case as one of cortical softening.

Dr. CAMPBELL, in reply, said that the patient had a moist skin and the hair was not coming out, so that he thought myxœdema could be safely

excluded. He did not incline to Dr. Guthrie's view as to the possibility of its being primarily of cerebral origin, the sclerosis being secondary. This view implied the existence of a symmetrical and progressive lesion limited to the motor tract in each hemisphere. He could think of no morbid process likely to account for the symptoms but a primary sclerosis.

Anæmia in a Man æt. 42.

Dr. CAMPBELL showed a man æt. 42, by occupation a hat salesman, the subject of pronounced anæmia. The patient was prematurely grey, and had had a great deal of mental trouble. He could find no cause for the condition; there appeared to be nothing in the way of malignant disease, nephritis, or Addison's disease to account for it. There was nothing in the blood to suggest pernicious anæmia or leucocythæmia. In reply to the chairman, Dr. Campbell said neither the spleen nor the other abdominal organs were palpable. There was no enlargement of glands and no history of vomiting. One must be content to call the case one of idiopathic anæmia. The pathology of such cases deserved careful study.

Cystic Degeneration of the Breast.

Mr. JACKSON CLARKE showed a specimen of a breast he had removed for cystic degeneration. He said the condition was of sufficient severity to demand amputation of the breast. The patient was a woman of 50, which was the usual age for the disease. Enlargement of the breast had only been evident to her for five weeks when she sought advice. At first he was inclined to regard it as a duct carcinoma. The breast tissue covering the cyst was thickened considerably, and the members would appreciate the difficulty of distinguishing a tense cyst from a growth. He thought the present case was one of duct cancer that had hæmorrhage into it, and thus drawn the patient's attention to it. That was the assumption which led him to advise removal. Before removing any breast, however, he thought it was imperative that the surgeon should make an incision into what appeared to be a growth, because a cyst or a chronic abscess might give exactly the same sensation to the hand as a neoplasm. On incising the cyst which he was exhibiting it was found to be filled with a greasy material; thereupon

the question arose, was this a simple cyst, or only part of a general cystic change throughout the entire breast? At the operation Mr. Clarke found that on squeezing the breast, although the cyst which had been evident before had been emptied, there was a discharge of creamy stuff from the nipple when the breast was pressed. The patient was past the climacteric period, so there was no question of lactation. Having examined other specimens like the present, he recognised the condition as one in which every part of the breast was becoming cystic. Was there any treatment short of amputation that was of any good for the condition? In a case in which Mr. Barker had removed such a breast, a fragment was by some means left behind, and this fragment some months later began to produce cysts on its own account, *i.e.* the condition recurred as if the growth had been carcinomatous. In another case which came to his notice, and in which he examined the breast histologically, every fragment of epithelial tissue was undergoing this proliferative change which led to the cystic degeneration. If a name were required for the condition it might be called diffuse adenoma of the breast, comparable to diffuse adenoma of the thyroid gland, or of the cervix uteri. If the breast were not removed the patient would become increasingly distressed owing to the great increase in size; discharge might occur from the nipple, and there was a possibility that the tumour might become cancerous. The disease of the breast of which he showed a specimen constituted a definite entity both pathologically or clinically, and required a pronounced mode of treatment.

Mr. MAYO COLLIER said he had never seen a breast in the condition which Mr. Clarke had related. Did Mr. Clarke wish the members to understand that there was no new tissue formation in the breast, and that the essence of the disease was simply a blocking of the tubes of the breast? In new growths of the breast nothing was more common than cystic degeneration. It was something new to him that a breast should simply degenerate into cysts without any new formation of any sort whatever. He agreed that the only course of treatment in the present case was removal.

Mr. AYDON SMITH said he would like to ask if occlusion of the ducts in infancy would not con-

tribute materially to the production of a condition of the kind under discussion. In confinement cases, particularly where an old-fashioned midwife was in attendance, the midwife delighted to knead the female child's breast until an abscess was formed, then on squeezing out the pus she imagined that it was milk. He had seen very little mention of the point in works on the subject, but attention had been recently drawn to the matter in the 'British Medical Journal,' though no reply had yet appeared to the letter. He (Mr. Smith) believed that this brutal treatment of breast at infancy had very much to do with after-trouble in the mamma.

Mr. TEMPLETON asked if the case might not be a hygromatous degeneration affecting the cellular and adipose tissues.

Mr. TURNER asked whether Mr. Clarke suggested there was any new growth of the mammary tissue. There was a reference in Erichsen's 'Surgery' to cystic degeneration of the breast, which was described as occurring after the climacteric, due possibly to mere interstitial constriction blocking the tubes and leaving unabsorbed portions of the ducts or acini, which latter enlarged from continuing their natural secretion. That there should be any fresh formation of mamma tissue he did not think was probable at that age.

Mr. JACKSON CLARKE, in reply, expressed his gratification at the interest his case had evoked. He thought attention had only recently been drawn to the condition very prominently, and that it was Mr. Barker's case which had aroused that interest, the case having been reported by Mr. Raymond Johnson. Mr. Collier's remark that the condition was very rare was true; simple galactoceles were also rare, and were supposed to be due to ducts having ruptured and the milk being diffused into the connective tissue. Retention cysts of the breast were very rare, but new growths with cysts were common. Another kind was called the serous cyst, because it contained serum and lay in a simple cavity lined with epithelium, probably in lymphatic spaces. A cyst which was allied to his case was an adenoma which sprouts, as well as that in which there was an ingrowing adenoma. In the condition he showed the breast was no longer a useful organ, for every part of it secreted fatty substance, and there was a progression of acini and the formation of cystic spaces. In

one specimen he had examined the ducts were blocked.

Referring to the remarks of Mr. Smith, he (Mr. Clarke) had seen an abscess in the breast of a child a few days old. The usual time for such inflammatory conditions of the breast to occur was about the eighth day of life and at puberty. Mr. Templeton had referred to an hygroma; that might be compared to a cavernous angioma—a nævus of the lymphatics instead of the veins. It could hardly be an hygroma, because no such matter came out of the breast on squeezing. He had examined a similar case to the one he was showing, under the microscope, and he knew the condition was due to the change in the epithelium and the acini; it was like what was found in adenoma, but there was no fibrous growth supporting the new glandular tissue.

Appendicitis.

Mr. CLAYTON showed a young man on whom Mr. Durham had performed an operation for appendicitis twelve months previously. Members of the staff of the hospital would remember that the case presented such diagnostic difficulties that a consultation was held. The consensus of opinion was that it was a genuine case of appendicitis. It was thought wiser to send him back to bed for a time before operating. At the end of two or three weeks the patient began to go downhill, his temperature rose, and there were other evidences of the formation of pus in the peritoneal cavity. The abdomen was therefore opened in the middle line, and when he came to the peritoneum there was a gush of most offensive pus, amounting to more than a pint. The wound was freely enlarged, the cavity washed out gently with boric lotion, and no attempt was made to do anything further. A drainage-tube was placed in and pushed down as far as possible, absorbent dressings being applied. The patient steadily got well, and left the hospital three months after the operation with a small sinus, which soon after completely healed. The first symptom the patient complained of was a feeling of indigestion, followed by pain in the epigastrium well above the umbilicus. This pain increased in severity, so that he was unable to get about. "McBurney's point" was found to be tender. He also had a peculiar symptom which he had not

read of in association with appendicitis, namely, retention of urine, apparently due to intense spasm of the deep urethra, which made passage of a catheter very difficult. There was also pain in the rectum, which he had noticed in some of these cases. The patient was now in perfect health, as would be evident to the members, the bowels acted well, and he was able to do a good day's work. He (Mr. Clayton) thought Mr. Durham was perfectly right to be content with opening the abdomen and washing out afterwards; searching for and attempting to remove the appendix in this case would have been a most dangerous and unjustifiable proceeding, and the present condition of the patient justified the course of not attempting too much. Moreover, experience showed that in most of these acute inflammations with abscess formation, the appendix became obliterated and caused no further trouble.

Mr. MAYO COLLIER said he believed that the patient was almost moribund when admitted, and that there was an enormous abscess cavity. It was not a question of removing his appendix, but one of preventing his being removed speedily to another world. The only thing to do was to open the abscess and drain, and leave the cavity to collapse and contract. To search for the appendix in such cases would be like the proverbial searching for a needle in a bundle of hay. Seeing the agglutination and adhesions which often took place in these cases it was remarkable that the patient had had no symptoms since the operation. These cases of appendicitis gave the practitioner and the operator more trouble than any other class of cases. He would like to narrate a case in which he did the wrong thing. It was an acute case in which there was a perforation and acute peritonitis. The patient five or six weeks ago had symptoms of jaundice, he had acute agony over the liver, with a tender area, and he therefore thought it was an abscess of the gall-bladder. He cut down and found the gall-bladder quite healthy; he, however, found general peritonitis, but at the end of the operation the patient was so bad that the proceedings could not be carried any further. That patient died of acute appendicitis. There was no history pointing to appendicitis. Post mortem the perforation was found causing general acute peritonitis. He had seen a great many cases of appendicitis, and in some of them it was

almost impossible to make out what was the trouble.

Mr. JACKSON CLARKE said he was sorry Mr. Durham was not present to witness the perfect success of his treatment of this case. Appendicitis was a subject of great interest to members of almost all walks of the profession. The best recognised classes of cases were—(1) appendicitis with diffuse peritonitis, (2) appendicitis with local perityphlitic abscess, and (3) relapsing appendicitis. In the first class of cases only a very early operation gave the least possible chance, so that from the earliest stage appendicitis should be closely watched to see if it was going to take that course. The case before them was really between the form he had just mentioned and those of the second group. In this patient examination of the rectum revealed a distinct induration just above the prostate, showing that pelvic peritonitis was present. Mr. Durham's decision to wait turned out to be a wise one. He would recommend to Mr. Clayton that a pad should be worn by the patient to prevent a hernia, as there was a distinct circular area of weakness at which a hernia might well form. Whenever a local perityphlitic abscess formed it should be opened in such a way as to avoid infecting the peritoneal cavity. In these cases there was no hard and fast rule as to the removal of the appendix; that must be decided after the parts had been exposed. In cases of relapsing appendicitis the vermiform appendix should be removed between attacks.

Cases treated by the Dry Hot Air Method.

Dr. KNOWSLEY SIBLEY showed three cases treated by the Tallerman-Sheffield dry hot air method. After explaining the apparatus, which had been in the hospital some months, he said that the treatment consisted in placing the limb into a cylinder containing very hot air often as high as 280° — 300° F., &c., and keeping it there nearly an hour every two or three days. The cases he showed illustrated the improved condition in the nutrition of the parts so treated. The first case was one of chronic ulcer on the leg of a man *æt.* 60, who came under his care suffering from general bronchitis and dyspepsia. The ulcers—two in number—had existed more than a year, and were situated just below the left internal malleolus, being about

one and a half inches in diameter. There was a good deal of general œdema of the part, and the leg was very painful. The patient was ordered some boracic ointment; and as the ulcers were getting larger instead of smaller he was ordered the hot-air baths, the first of which he had on February 2nd. After two baths it was obvious that the ulcers were healing, and the healing was complete on March 1st, by which time the patient had had ten baths. The general nutrition of the whole limb had also considerably improved, there was less œdema, and the inflammatory area around the ulcers had disappeared. He considered that this hot-air treatment compared very favorably with the oxygen treatment for ulcers and other diseases. The second patient had suffered from malnutrition of the leg after ligature of the femoral artery and injury to the nerve. He was in the hospital under Mr. Durham from September, 1895, to July, 1896, with an abscess in the popliteal space which eroded into the popliteal artery. This was ligatured, and afterwards the femoral had to be tied on account of hæmorrhage. There appeared also to have been considerable injury to the popliteal nerve. When he first came under Dr. Sibley's treatment in October of last year, the leg was cold, very blue, and much withered. There was considerable impairment of sensation in places, especially about the foot, complete anæsthesia of the great toe and inner side of the foot; there was also perversion of sensation, the ankle was very stiff, and the patient could hardly walk. By March 15th the patient had had eighteen baths. The limb was much stronger, and he was able to get about comfortably; the feeling in the foot was much improved, and the limb remained constantly warm. The general nutrition of the part had also improved. The third case was one of malnutrition of the hand due to severance of the nerves of the wrist. The patient received a severe cut across the wrist-joint in October last year, which was followed by loss of sensation in the last three fingers, and the hand could not be used for anything. The first bath was given on January 4th, and the patient stated that some feeling returned after the second bath and remained for a few hours, but was lost again afterwards. On February 16th he had had six baths, and was able to use his hand a little. Sensation had also returned to a considerable degree in the fingers.

Mr. JACKSON CLARKE agreed that, like massage, hot-air baths had the property of improving the nutrition of particular parts. One of the patients exhibited he noticed had some degree of equinus, and he recommended a small operation being done when the limb was sufficiently well nourished for the patient's future comfort. The hot air treatment was a valuable adjuvant to surgical treatment.

Cervical Auricle.

Mr. TEMPLETON showed a boy from the wards, with a cervical auricle on the left side only. There was a slight depression of the skin, but no sinus, and no mucus escaped. Its position corresponded to the second branchial cleft, and it contained a nodule of cartilage. He believed one other member of the same family had a similar deformity.

Mr. J. G. TURNER said, in animals, teeth had been observed with cervical auricles and persistent branchial clefts, as in a specimen from a sheep's neck, probably of persistent second branchial cleft, preserved in the Museum of the Royal College of Surgeons, London. The temporal tooth of the horse was an example of dental structure developed in connection with a persistent cleft; there was usually a small sinus leading down to such a tooth, though there had been no preceding suppuration. Cervical auricles probably represented the opercula of fishes.

Mr. SWAINSON asked if cervical auricles were not usually bilateral.

Mr. TEMPLETON, in reply to observations, said that macrostomia might be associated with auricular appendages, such as he had seen in one case at the Children's Hospital, Great Ormond Street, and Mr. Morgan had reported another, in which macrostomia was associated with supernumerary auricle. He further remarked that such deformities had a tendency to be bi-lateral, and to occur in successive generations, quoting a case where cervical auricles were present in grand-parent, parent, and child.

Chocolate Emulsion of Cod-liver Oil.—Chondrus mucilage, N. F., 5 oz.; Cod-liver oil, 8 oz.; Glycerin, 2 oz.; Cocoa, powdered, 1 dr.; Vanilla tincture, 3 dr. Rub the cocoa with the mucilage, and heat until a uniform mixture is obtained. When cold, add the cod-liver oil and glycerin, and beat up with an egg-beater.—*Practical Druggist.*

NOTES.

Deep Lesions produced by the Roentgen Ray.—It is seldom that an issue of the 'Bulletin' of the Johns Hopkins Hospital does not contain at least one paper of unusual merit and originality. The current article has for its *pièce de résistance* an admirable illustrated article on the effects of the X ray upon osseous structure, by Mr. T. C. Gilchrist, Associate in Dermatology at the Hospital. The author finds twenty-two recorded cases of superficial effect, but his own is the first in which the deeper effect of the X ray have been demonstrated by a radiograph. His patient, a male, was an X ray operator, the eruption or dermatitis occurring on the dorsal surface of the right hand, wrist, and lower forearm, after frequent and long-continued exposures. There was hyperæmia and swelling of back of hand, inflammation of hand and fingers; the integument became dark brown in colour and later exfoliated. There was no pain at first, but later the pain was so severe as to cause the patient to stop his work. There were aching, shooting, and throbbing pains. The bones of the hand were very tender on pressure, particularly the first phalanges of the index and second finger and the carpal bones. The movements of the hand became so limited that it was practically useless for some weeks. Sensation was also much impaired, but after exfoliation occurred it recovered again, but only gradually. Recovery of all the lesions has been very gradual indeed.

The photographs have revealed what has never been observed before, viz. a distinct osteoplastic periostitis, and probably an osteitis, particularly of the first and second rows of phalanges of the index and second fingers, also of the heads of metacarpal bones of the same fingers, and, judging from the symptoms, even of some of the carpal bones.

This then accounts for the severe symptoms, the aching, throbbing, and shooting pain which prevented sleep. The density of these bones also has been increased, showing that even bone tissue has been affected.

A complete demonstration is thus afforded of the powerful, piercing character of the X rays, and the severe painful symptoms which have been described by other observers are probably due to the

inflammation of the periosteum, and possibly the bone, besides the softer tissues.

This inflammation has also extended to joints, which would explain the loss of movements, and pain, when they could be used later.

As a result of these observations, it proves that the X rays are even more powerful than have been generally thought, that the deleterious effects can in some cases be quite serious, and that the cutaneous manifestations are not the most severe of the lesions, but those of the deeper tissues, and particularly of the periosteum and bones, being more severe.

He consulted with Professor Ames, Associate Professor of Physics in the Johns Hopkins University, who, after reviewing all the facts of the case, kindly wrote as follows concerning the present theory of the X rays:

"The radiation in an 'X-ray tube' may be divided provisionally into three classes: ether waves, which may have wave lengths from 150 to 800 mm. approximately; cathode rays, which undoubtedly are streams of matter, electrically charged; X rays, about whose nature there is no conclusive evidence at the present time. If the walls of the tube are thin enough and of suitable material, all these radiations will emerge and pass into the surrounding air. It is a matter of doubt if the cathode rays observed outside the vacuum tube are the same as those inside; but the inner ones undoubtedly cause the outer ones. There is no evidence that X rays carry with them particles of matter, or that they directly cause a stream of particles; in fact, all known facts seem to point to the belief that they are ether waves of extreme shortness."

It will thus be seen that the opinion expressed here does not make it possible for the X rays themselves to produce such deleterious effects as have already been described, but Dr. Ames mentions the fact that the cathode rays are undoubtedly streams of matter electrically charged. Here then we have some possible grounds for the theory that the lesions may be due to the entrance of particles (platinum in our case) into the injured tissues, and that the cathode rays which accompany the X rays may be the cause of the trouble, and not the X rays themselves.

On clinical grounds there is considerable support for this, at first sight, improbable theory.

If the lesion extends at all deeply, it leads to the formation of ulcers which are extremely intractable, and they may be due to irritating particles still present in the tissues.

We do not think that the possibility of injury ought to deter one from using these wonderful rays in surgical work, because only a few have been affected out of thousands who have been exposed to them. By keeping, as Thomson says, some distance away from the rays, injurious effects will hardly follow their use, and when the exposure is for a short time, unless, as may happen in all other diseases, idiosyncrasy plays a prominent part.

When the hand was viewed two or three times near a new Edison bulb, through a tungstate of calcium screen, after four or five minutes a tingling sensation was distinctly felt throughout the dorsum of the hand; this symptom lasted for ten or fifteen minutes, and passed away without any further results. It occurred to him that X ray operators and experimenters should expose to the rays the palmar surface of the hand, which is protected by a much thicker horny layer, rather than the back of the hand, which is much less protected.

In conclusion, we would strongly advise all X ray operators and experimenters who develop any special idiosyncrasy, to abstain from their use if they find that the slightest deleterious results follow an exposure to them.—*Journ. Amer. Med. Assoc.*

Gout.—Dr. Hans Froelich ('*Journ. of the Amer. Med. Assoc.*,' January 30th) thinks that local treatment is the most effective. We are accustomed to regard a surgical treatment of the local precipitation of urates, the tophi, as almost a sacrilege. However, they are foreign bodies, and all foreign bodies, if accessible, ought to be removed. There is much talked and written about dissolving these urates with lithium salts, piperazin, and, of late, lysidri. They work nicely in the reagent tube with water; but as soon as we take urine the dissolving effect stops. Dr. Froelich is unable to say how they work in plasma. If they are locally injected in a solution strong enough to take effect, the resulting chemic process causes a painful inflammation which lasts almost two days. The only way to remove these tophi is by curetting under asepsis; it ought certainly to be done in each case during the time no acute attack is apparent.—*Med. Rec.*

The Surgery of the Chest. By STEPHEN PAGET, M.A.Oxon., F.R.C.S., Surgeon to the West London Hospital and to the Metropolitan Hospital. Illustrated. (John Wright & Co., Bristol. Price 10s. 6d.)

IN the preface to this volume the author says that "there are signs that we have reached a stage, in this portion of our art, beyond which, on our present lines, we cannot advance much further." This statement justifies the publication of the present work on the surgery of the chest. The book is the first of its kind that has been published in English, and is the outcome of much patient investigation. There is one point which is very marked in this publication, and that is the great modesty of the author in stating his own views on the subjects treated. About one third of the book is devoted to injuries of the chest, and in the remaining two thirds the treatment of such affections as are suitable for operative interference meets with adequate and thorough explanation. The value of the work to the ordinary practitioner is very real, and one need only refer to the treatment of foreign bodies in the air-passages to be convinced of the usefulness of this volume to those engaged in active work. Referring to fracture of the sternum with irreducible displacement, cutting down on to the fragments and wiring is discussed. Dealing with pneumothorax, the necessity for an early operation is insisted upon. In regard to wounds of the intercostal arteries, it is pointed out that if the hæmorrhage is primary, the wound should be plugged; but if secondary, resection of the ribs should be performed and the vessel secured. In treating of the wounds of the heart, the number of cases in which recovery has occurred is certainly encouraging. The question of aspiration is sufficiently discussed, and the inhalation of oxygen is recommended for the treatment of acute œdema of the lung with albuminous sputa—a condition which may follow too rapid aspiration of a pleuritic effusion. Empyema is discussed in a very complete manner, the essential points are emphasised with much skill, and the fact that simple incision in many cases is held to be sufficient, is accorded a just and proper acceptance. Particular attention has been given to the consideration of operating for abscess in the lung, and the advantages are carefully set out. With a limited space it is impossible to do justice to a work of this excellence; in fact, to fully appreciate its scholarly style and scientific accuracy, the book must be not only read, but studied.

THE CLINICAL JOURNAL.

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ON SOME PAINFUL AFFECTIONS OF THE FEET.

By A. H. TUBBY, M.S.Lond., F.R.C.S.Eng.,

Assistant Surgeon to, and in charge of, the Orthopædic Department, Westminster Hospital; Surgeon to the National Orthopædic Hospital; Surgeon to Out-patients, Evelina Hospital for Sick Children.

PART II.

Acquired Flat-foot in Adolescents and Adults.

Acquired flat-foot arises from paralysis of the peronei muscles, from rickets, from injuries, especially after Pott's fracture, and from spastic conditions of the central nervous system; and there is the variety known as the painful flat-foot, of which more anon. The importance of recognising rickets as a cause of flat-foot is that, that if these cases are left untreated, they develop later on into the most inveterate painful forms of flat-foot; for instance, in a rickety child the following stages may be seen:—*Pes planus*, or simply the dropping of the arch without any eversion of the foot, and then what is known as ingrowing ankles or relaxation of the ligaments of the ankle; and finally, true flat-foot, that is dropping of the arch of the foot together with eversion of the foot and abduction of the same. Most varieties of talipes valgus are not accompanied by pain, except such as is due to pressure on displaced bones; but in the flat-foot of adolescents and adults one of the chief symptoms is pain, and I now propose to describe somewhat fully the painful flat-foot and its treatment.

The Degrees of Flat-foot.

First degree, or the oncoming flat-foot. There is seen some sinking of the arch when the patient stands, and is told to bear the weight fully on the foot. This sinking disappears on adduction of the foot, standing on tiptoe, and on sitting. Pain is frequently present at this stage.

Second degree, or pronounced flat-foot. The arch

has sunk to a considerable extent, although the head of the astragalus is not touching the ground. The deformity cannot be reduced by any effort of the patient, nor can the feet be voluntarily inverted; the peronei tendons and the extensor communis digitorum are felt in relief, and there is considerable muscular spasm and pain.

The *third degree*, rigid or spasmodic flat-foot. The astragalus and scaphoid are now touching the ground, the foot is very tender, the patient can only hobble about, and the deformity can be reduced neither by the patient nor by the surgeon. The peronei and the extensor communis digitorum are in strong relief, and the thickening of the soft

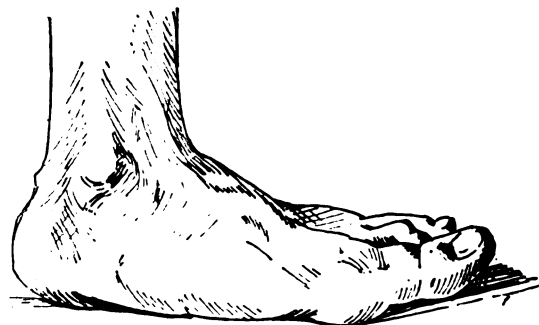


Fig. 12.—Inside view of a case of severe flat-foot (third degree).



Fig. 13.—Outside view of the same foot as in Fig. 12.

tissues on the inner side of the foot is much in evidence. The eversion and adduction of the anterior segment of the foot are very noticeable (Figs. 12 and 13).

The *fourth degree*, or osseous flat-foot. Of this degree I have met with but few cases, but they appear to have been seen frequently in the practice

of Professors Stokes, Ogston, and others. The foot is rigid, board-like, very tender, and extremely distorted, and some of the joints are ankylosed.

Etiology of Flat-foot.—In the majority of cases there are three factors involved, namely, adolescence, feeble health, and strain on the feet out of proportion to the muscular development.

Adolescence.—Mention has already been made of the occurrence of flat-foot in the rickets of childhood. Either owing to the persistence of post-rachitic weakness of the ligaments, or to the occurrence of late rickets, flat-feet are sometimes seen in childhood. At the time of puberty, especially in girls, the body becomes weighty, and at the same time the general health is often impaired by the strain incidental to that period. At this age, too, the foot often becomes unduly lengthened, the arch is therefore extended, and it is these long feet which are very liable to become flattened and to assume rapidly a severe form.

Feeble or Impaired Health.—Under this heading there is weakness following the exanthemata; one of my cases suffered from scarlet fever at five, measles at eight, succeeded by pneumonia, and a second attack of pneumonia at twelve years of age, and the flat-foot began to appear at thirteen. Acute rheumatism is responsible for some cases. Excessive strain on the feet, especially some occupations involving long standing on weak feet (static club-foot). Thus amongst my cases some of the occupations were grocer's assistant, gardener, butcher, errand-boy, porter, barman, waiter, domestic servant, and nursemaid.

In addition to these general factors there are other local conditions,—for instance, shortness of one limb; in this case progression is easier if the inner side of the foot is brought to the ground first. High-heeled and narrow boots often result in flat-foot; so, too, stiff or ungainly boots produce flat-foot by preventing sufficient play of the muscles of the leg and foot. Another local cause is gout. Such an instance occurred in a woman aged forty-nine; eighteen months before being seen by me she had an attack of gout in the right foot, which is now in the second degree of flatness.

Injury.—Excluding examples of badly set Pott's fracture, which often become valgoid subsequently, it is a common observation that the date of onset of flat-foot coincides with a sprain or injury, which

in itself is insufficient to account for all the deformity. I saw, in 1891, a boy aged eight years whose right foot had been run over three months previously, and who developed a painful, rigid condition in that part.

Pathology of Flat-foot.—Abduction of the foot is the position of weakness, and adduction of strength and activity. Bradford and Lovett remark that "flat-foot is the result of a disproportion between the body-weight and the apparatus for sustaining it." Professor Sayre holds the view that flat-foot is due to paralysis of the tibialis anticus. With reference to this I may say that although, in my opinion, it does not explain the majority of flat feet, yet I have seen at least three cases in which a distinct groove could be felt along the outer side of the crest of the tibia for its whole length, and



Fig. 14.—Severe flat-foot. The figure shows the groove on the outer side of the crest of the tibia, due to wasting of the tibialis anticus.

corresponding to the position of the tibialis anticus (Fig. 14). In these cases, although the muscles were not paralysed, they had undoubtedly undergone some wasting.

Morbid Anatomy.—Changes are seen in all the structures of the foot, but chiefly in the ligaments; the most marked changes are in the inferior calcaneo-scapoid and the calcaneo-astragaloid ligaments; the former is relaxed and broadened, and

has resting on it more of the anterior aspect of the head of the astragalus than is normal. Of the calcaneo-astragaloid ligaments the interosseus is stretched, and its fibres are separated one from another. The external ligament of this articulation is also longer than naturally. As the ankle becomes more valgoid its ligaments suffer in proportion. The superficial part of the internal lateral ligament is elongated and thinned, while its deeper part suffers but little. The external lateral



Fig. 15.—Complete cure of the foot seen in Fig. 14.

ligament shows changes chiefly in its middle fasciculus, which is thinned and atrophied (Fig. 15).

The Fasciæ.—In early cases, just as the arch is giving way, I have felt the inner band of plantar fascia very plainly in relief. In such examples it is probable that the ligaments and muscles have yielded before the fascia.

The Muscles.—In advanced cases the leg loses much of its roundness, owing to gradual wasting of the muscles.

The Bones.—Various changes take place in the position of the bones; the astragalus is displaced downwards, forwards, and inwards, while the bones in front of the astragalus and os calcis are displaced upwards and rotated outwards (see Figs. 16 and 17).

The Malleoli.—The external malleolus in severe cases is in the same transverse plane as the internal,

or even in front of it, while the position of the internal is but little altered.

Symptoms.—They are—1, pain; 2, swelling; 3, flattening of the sole; 4, alteration in gait; 5, loss of shapeliness of the foot; and 6, congestion and sweating of the feet.

1. *Pain.*—In many instances this is present from the first. Occasionally it is complained of only after a twist or sprain of the foot. In most cases of flat-foot of severe degree pain is a prominent symptom. Occasionally it does not happen to be present. The character of the pain is not uniform. In the early stages there is at first a feeling of fatigue, succeeded after a few days or weeks by a dull aching which is noticed in the feet, and then extends to the legs and even to the thighs. On resting in the evening with the feet raised the aching passes away, but as the daily occupation is persisted in the aching comes on earlier in the day, and towards evening assumes a sharp and intense form, shooting up into the legs and thighs. In the early stages of this sharp pain a night's rest is followed by freedom in the morning, but after a time there is a considerable loss of sleep from it, and when the patient awakes he finds the feet stiff. On rising and placing the feet on the ground the aching at once recommences, and finally the patient is unable to stand for any length of time, and can only hobble with considerable difficulty.

The tender points in the feet are well defined and very sensitive to pressure; they are found beneath the head of the astragalus and the tuberosity of the scaphoid below and in front of the internal malleolus, also on the dorsum of the foot just in front of the ankle, and about the bases of the first and fifth metatarsal bones. Less often pain is felt about the external malleolus, and on the outer side of the shin and up to the thigh. The causation of pain is as follows:—The feeling of fatigue first complained of arises from overwork and stretching of the muscles; they have not sufficient vigour to repair the metabolic changes of ordinary daily wear and tear. The dull pain is due to stretching of the ligaments and fasciæ, and the pull upon the enfeebled muscles. The sharp pain has its origin partly in the continued strain on the soft tissues; more often it is referable to surface of bone which, not normally in contact, are brought into relationship with one another at points not accustomed to

pressure. Arthritic changes explain some of the worst degrees of pain, especially when the latter is situated at the base of the first metatarsal bone; here distinct hypertrophy is often present.

2. *Swelling of the Feet.*—Local puffiness is often seen over the tender points, and redness from time to time dependent on the amount of standing and walking. When redness is present the mere touch of the finger increases the pain. If the case be of rheumatic origin the pain is worse at night, and effusion into the tendon sheath occurs. In old-standing cases, especially if varicose veins are co-existent, a general œdema of the foot occurs. In cases of the third degree corns and false bursæ, or bunions, form over prominent portions of bone. Inflammation of these adventitious structures is attended with exquisite pain.

3. *Flattening of the Sole.*—The existence of this is best obtained by taking a tracing or outline of the tread; thereby the slightest cases ought not to escape recognition, and the degree of improvement or the reverse can be noted from time to time.

4. *Alteration in the Gait.*—In cases of some severity the gait is lumbering and awkward; the patient is splay-footed; he walks with considerable difficulty, and cannot move quickly. The foot is no longer elastic, and walking is further impeded by the attendant pain. The loss of elasticity is due to the stretching of the ligaments and alteration in the position and direction of the bones, especially of those entering into the mediotarsal joint, and to a less extent of those at the ankle-joint. The foot is kept stiff and rigid, and the patient appears wooden-footed.

5. *Loss of Shape.*—This is noticed by the practised eye at once, and needs no full description.

6. *Sweating of the Feet.*—By many flat-feet and sweating are said to frequently co-exist; the explanation may be that both are associated with feeble local innervation of the vessels and other structures.

Prognosis.—Flat-feet do not become cured without treatment. As a rule the pain and disability become steadily worse, and the patient has to seek relief from the inconvenience thereby occasioned. Occasionally after several years, when the breaking down of the arch has ceased, the pain disappears, but the foot is permanently damaged, and has lost nearly all its usefulness.

Diagnosis.—Probably there is no deformity so easily and so often overlooked as slight acquired valgus; but a careful examination of the foot, especially if a tracing of the sole is taken, will often reveal the true cause of the pain and swelling. I have known cases to be treated for rheumatism, gout, and for otitis of the bones of the tarsus, but as Bradford and Lovett justly remark,



Fig. 16.—A case of spasmodic flat-foot, showing prominence of the internal malleolus and tuberosity of the scaphoid.



Fig. 17.—The same foot as in Fig. 16, as seen from the outer side. The elevation of the outer border and eversion of the front part of the foot are well shown.

there is no need for entering upon any elaborate and differential diagnosis, for mistakes are oftenest the result of carelessness. It is a wise precaution to investigate the condition of the arch in all painful feet (Figs. 16 and 17).

Treatment of Painful Flat-foot.—This must be both general and local.

General Treatment.—If anæmia is present, iron should be given for a considerable period. Change of air and rest are often urgently called for. When the rheumatic diathesis is present, salicylate of soda in acute and subacute cases, and in the chronic, iodide of potassium and tincture of guaiacum will often be found serviceable. Gonorrhœal rheuma-

tism is very intractable. Iodide of potassium and cod-liver oil are said to do good. When gout is associated with flat-foot, citrate of soda, citrate of lithia, and piperazin, associated with suitable dietetic treatment, will ameliorate the general condition. In rickety flat-foot, cod-liver oil, phosphate of iron, and plenty of fresh milk and pure air will go far to effect a cure. The relief of pain is often a pressing necessity. The surest therapeutic measure is rest, entire and absolute. But unfortunately patients rarely understand the full value of rest, and indeed are often unable from force of circumstances and surroundings to obtain it. At the most the rest is only partial. If the feet are acutely sensitive, the application of extract of belladonna and glycerine, or warm opium lotion, or poppy fomentations give temporary relief. Rébard suggests the injection of cocaine near the painful joint, but it is scarcely advisable to habituate a patient to the use of this drug in so chronic an affection as flat-foot.

Local Treatment.—The means which can be adopted are rest, exercises—passive and active,—mechanical support, operative measures, including forcible manipulation; but the exact line of treatment must depend upon the degree, severity, and duration of the deformity. Briefly, it may be said that in the first and second degrees of flat-foot, passive and active exercises and supports will all be employed, but each to an amount varying with the special causes at work, and the condition of the muscles and ligaments. To illustrate my meaning I would instance the case of flat-foot of the first or second degree in an anæmic overgrown girl. It is useless to trust to tiptoe exercises as an immediate remedy; the patient is physically incapable of raising herself on tip-toe, and if she do so the strain on the muscles and ligaments is so great as to cause further breakdown of the arch. This class of case requires rest and support as the prime factor in the treatment; and when the parts have regained their position the muscles may be actively exercised, so that the best means are taken to prevent a return of the trouble.

Treatment of the First and Second Degrees.—As Mr. Golding-Bird remarks, the principle of all successful treatment in acquired valgus is to relieve the overstrained adductors of the foot, that they may recover their tone, offer due opposition to the peronei or abductors, relieve the pronation and the

eversion, and raise the sunken arch by giving elastic support upwards and inwards. Before this can be done all active spasm on the part of the peronei must have ceased. In static and rachitic cases of this degree the first essential is absolute rest, and with this may be combined the relief of the abduction, thus: the patient should be told to sit on a comfortable sofa or bed, tailor fashion, that is with the legs crossed, with the weight of the limbs bearing on the outer side of the foot. By making use of this posture, which is maintained for from two to four weeks, the foot rapidly regains its normal appearance, the pain ceases, the arch rises, and the deformity is temporarily relieved. But rest merely allows the foot to return partially to its normal position, and gives the exhausted muscles and weakened ligaments breathing-time, so to speak. Then two other methods should be employed in addition, viz. exercises and an elastic support to the arch of the foot, the exact proportion of the use of each being dependent on the general and local conditions.

Exercises.—Those most to be recommended are the tiptoe movements. The object is to strengthen the flexors of the toes, especially the long and short flexors of the great toe. At the same time the tibialis posticus should be called into vigorous action, and with the extension of the foot the tibialis anticus comes into play. The exercises should be carefully regulated. The patient stands erect with the arms by the side. The feet are placed on the ground somewhat adducted and inverted, the toes being nearer together than the heels, and the knees fully extended. The patient then raises himself on tiptoe, and immediately resumes the position of rest. It is best to do this in rhythm to the swing of a pendulum, or to the beat of a metronome, which can be set to the required rate. At first two to three minutes' exercise twice a day is all that can be borne, the patient ceasing the exercise before pain or fatigue is felt. But as the muscles acquire strength and vigour, the duration and the number of times a day may be increased. The same idea of exercise is carried out by going upstairs, first bringing the toes into contact with the stair and then the whole foot. Another form of exercise is to place the feet nearly touching, and to invert the soles so as to bear the body-weight on the outer edge. Then the original position should be resumed. This

last exercise may be done as often as the tiptoe exercise, and in place of it. Passive exercises, which are very valuable, should be carried out as follows:—The attendant takes the foot and performs a combination of extension movements at the ankle with rotation of the medio-tarsal joint, at first inwards and then outwards, but mainly inwards. Later, the patient should be able to perform them for himself.

Supports.—In very slight cases it is sufficient after resting to have the heel of the boot prolonged forwards on the inner side, and thickened at the inner edge. In the first and second degrees of flat-foot it is worthy of notice that the pain is immediately relieved by temporarily wedging up the inner border of the foot with a thin book or a piece of paper suitably folded; in fact, this is a good means of diagnosis in doubtful cases.

In many cases of the first and second degree of flat-foot I recommend the following arrangement of boot. The sole and heel should be one quarter of an inch thicker on the inner edge than on the outer edge, the thickening being gradually bevelled off so as to cease halfway across the sole and heel. The heel should also be prolonged forwards on the inner side, so as to come well beneath the arch of the foot; the waist of the boot should be strong and thickly padded; and in cases in which there is great muscular relaxation and a good deal of flaccidity of the arch it is well to insert a vulcanised rubber pad beneath the arch.

Pads and Surgical Soles.—Pads are made of various materials, viz. felt, wool, leather, steel, rubber, or hollow and filled with glycerine. Of these the vulcanised rubber are the most useful, and in the end the least expensive, since most other materials become sodden with perspiration and hard to the foot, while rubber becomes soft and more elastic with wear. To be of any value a valgus pad must be made a part of the boot. A good combination for the second degree of flat-foot is a boot with a vulcanised valgus pad and concealed spring, the spring being placed on the outer side of the boot, and so arranged that when the boot is laced up the foot is pressed inwards. In severer cases a boot with a valgus pad and an outside support to the knee with a valgus T-strap is indicated, the action of the T-strap being to lift up the arch of the foot. The surgical sole, which

resembles a cork sole with a steel piece shaped to the concavity of the arch, is of little or no value. A very excellent contrivance is Whitman's brace, made of steel, or preferably, since this rusts, of aluminium, which is light and comfortable, and does not interfere with the movements of the foot or muscles. At the same time the brace should fit so perfectly that there is no tendency for the foot to slide from the brace when the body-weight bears on it. These braces have been extensively used of late, in most cases with success, but where failure has attended their use on account of the pain produced, it has been due to the fact that the right moment for using the brace has not been selected. The arch of the foot must have been restored by manipulation in the first place, and the muscles have been given time to regain their normal tonicity. The application of the brace is then of value, since it tends to maintain the arch in a good position and to secure some inversion of the foot; but to attempt, as is often done, to apply a Whitman's brace to a flat foot in which the bony prominences are much in evidence, and the arch is not restored, is to court failure. The patient cannot wear the braces on account of the extreme pain produced by the pressure of the displaced bones against the firm steel or aluminium support. Another method of value is by elastic traction, which is best carried out by an arrangement suggested by Mr. Golding-Bird in the 'Guy's Hospital Reports,' some years ago.

Treatment of the Third Degree, rigid or spasmodic flat-foot.—The causes of the rigidity are two, viz. the altered position of the bones, and the pain and tenderness incidental to this stage, which set up reflex contraction of the peronei and extensor longus digitorum. These muscles may be permanently shortened or contractured, and the peronei tendons are sometimes dislocated forwards. But it is not always possible to be sure how much of the tightness of the peronei is reflex and how much is permanent. Before any division of these tendons is decided upon, it is well to take the patient completely off the feet for a fortnight. So much of the peroneal contraction may disappear that it is not necessary to perform tenotomy of these muscles. In the event of the peronei remaining contracted after a few weeks' rest, tenotomy should be done. In addition to the peroneal tendons, the tendo Achillis may need division.

Cases of this degree may be treated in two ways, either by forcible rectification of the foot under an anæsthetic, and then retention of the foot in plaster, or by the gradual methods, viz. tenotomy, passive exercise, and the employment of some apparatus.

Forcible rectification may be carried out either by the hand or by Thomas's wrench. If the hand be employed the patient should be fully under ether in order to relax the muscles. The surgeon stands in front of the patient, and, grasping the anterior part of the foot with both hands, the thenar eminence of his right hand, if it be the patient's left foot, and the reverse if it be the right foot, pressing firmly on the head of the astragalus steadily adducts and inverts the foot at the medio-tarsal and sub-astragaloid joints until the adhesions are felt to yield. Then rotation and circumduction movements are made in these joints, and finally the ankle-joint is forcibly moved in all directions. The effects noticeable are that the foot has come into better position at once, the peronei tendons are no longer tense, and the arch is partially or entirely restored. A thick flannel or cotton-wool bandage is applied nearly to the knee, then a plaster band is put on from within outwards in figure-of-8 fashion. Anæsthesia is maintained until the plaster is dried, so that the foot remains fully adducted and inverted and lightly flexed. Afterwards the patient may be allowed to walk, if extra strips of plaster bandage are applied to the inner side and to the heel. The bandage should be worn for a month. The same measures may be carried out with Thomas's wrench in the place of the hands.

The gradual method is as follows :—Division of the peronei and extensor longus digitorum tendons and occasionally of the Achilles tendons. An external malleable iron splint is then put on till the punctures are healed. The foot is readily got into position by means of Adams' modification of Scarpa's shoe. At least once a day, but better twice a day, the shoe is removed, and passive exercise is resorted to.

Treatment of the Fourth Degree.—Numerous forms of operations are practised for this degree, but the best of all is Sir William Stokes's. An incision one and a half inches in length is made below and parallel with the tendon of the tibialis

posticus over the most prominent part of the astragalus. A second incision joins this at right angles, and the small flaps thus made are turned back. From the neck and head of the astragalus a wedge of bone is taken away, and the wound is closed and the foot put up, according to the inventor of the operation in a Dupuytren's splint. Plaster answers equally well. The operation is only suitable in cases in which there is extreme rigidity of the foot, much bony distortion, and some ankylosis of the various joints. The result of the operation is to bring the centre of the astragalus within the so-called Meyer's triangle.

(To be continued.)

ON THE ÆTIOLOGY, PATHOLOGY, AND CLINICAL VARIETIES OF MALIGNANT ENDOCARDITIS.

BY

ARTHUR G. PHEAR, M.D., M.R.C.P.,

Assistant Physician and Pathologist to the Metropolitan Hospital.

IN TWO PARTS.

PART I.

MALIGNANT or infective endocarditis is one of the group of pyæmic diseases. It is characterised locally by an infective inflammation of the lining membrane of the heart, and is attended by general symptoms, more or less severe, of pyæmic nature. It differs essentially from simple or non-infective endocarditis in its origin, in the character of the local lesions, and in the nature of the general symptoms with which it is associated.

Ætiology.—The various diseases which may become complicated or followed by malignant endocarditis include many that are of septic nature from the outset, and others that, though not originally septic, are peculiarly liable to the super-vention of septic trouble. In a large number of cases of malignant endocarditis some or other septic antecedent can without difficulty be traced. The disease may arise in connection with suppurating wounds, puerperal fever, acute necrosis of bone, middle-ear disease, and like conditions. It may also occur in relation to lobar pneumonia,

and various acute specific fevers, such as scarlet fever and smallpox. Less commonly it arises subsequently to typhoid fever and diphtheria. It may occur also in conjunction with meningitis, but in such cases pneumonia is often present, and probably forms the connecting link between the two diseases. In gonorrhœa a form of endocarditis may arise which, apart from the associated condition, cannot be differentiated clinically or pathologically from malignant endocarditis due to other causes.

It is noteworthy that of these diseases some are attributable directly to the presence and growth of pyogenic organisms, while of the remainder nearly all are attended by special facilities for infection by such organisms. In scarlet fever and diphtheria pyogenic organisms may gain a footing and may flourish in the tissues of the fauces; a similar infection of the intestinal lesions may take place in typhoid fever. There is, no doubt, a possibility that the specific micro-organism of typhoid fever or of diphtheria may be the direct cause of the endocardial affection; and, indeed, the bacillus of typhoid fever has been detected in the vegetations when malignant endocarditis has followed this condition. But such cases are uncommon, and even in these it is impossible to make sure that pyogenic organisms have played no part in the production of endocarditis. There is, indeed, good reason for supposing that the cases are very rare in which pyogenic organisms are absent, and for regarding these organisms as an almost constant feature of the cardiac lesions.

A primary form of malignant endocarditis is said to occur, in which no indication can be obtained of any pre-existing condition to which the disease can be reasonably regarded as secondary. Such forms are at best extremely uncommon, and there is, moreover, always the possibility that some slight source of septic trouble has been overlooked. However careful and diligent the examination, the primary lesion may be so insignificant as to evade detection, and thus the endocarditis may have the appearance of a protopathic disease.

Acute rheumatism has little or no direct influence in the causation of malignant endocarditis. No doubt valves which are damaged by rheumatic endocarditis, whether the damage be of long standing or due to recent inflammation, are weakened in their resisting power, and are more

liable to become involved in processes of an infective nature than are healthy valves; but, except thus indirectly, rheumatic fever plays no important part in the ætiology of the disease.

To summarise the important ætiological factors:

1. Primary or protopathic malignant endocarditis, if it ever occurs, is of extreme rarity.
2. Malignant endocarditis may follow many of the acute specific fevers, including among these lobar pneumonia. Scarlet fever, smallpox, and pneumonia are more frequent antecedents than are diphtheria and typhoid fever.
3. The disease has a close relation to septic processes, and may originate in association with various forms of suppuration, puerperal fever, acute necrosis of bone, &c.
4. Valves that are permanently damaged from previous inflammation are more prone to be involved in an infective endocarditis than valves that are structurally intact.

Micro-organisms.

Many kinds of micro-organisms have been found and described in connection with malignant endocarditis. The more important are micro-organisms of the pyogenic group, and these probably play a leading part in the progress, if not in the production, of the cardiac lesions. The *Staphylococcus pyogenes aureus*, the *Streptococcus pyogenes*, and the *Diplococcus pneumoniae* are the three organisms most commonly found in the vegetations or ulcers. The diplococcus of gonorrhœa has also been detected; but no single one of these is constantly present, and the evidence goes to show that malignant endocarditis is not a specific disease, inasmuch as it is not caused by the presence and growth of any one micro-organism. There is, however, ground for believing, although it has not hitherto been proved, that the disease is closely associated with pyogenic organisms as a group. Even when other organisms are present (and the tubercle bacillus, the bacillus of diphtheria, and the bacillus of typhoid fever have been found in the cardiac lesions), one or other of the pyogenic organisms is also present, at any rate in a large majority of cases, and possibly in all.

Inoculation experiments have so far failed in that valvular lesions have not been produced by the introduction of micro-organisms into the blood so

long as the valves be uninjured. When, however, the valves have been previously damaged, mechanically or otherwise, the inoculation of pure cultures of the *Staphylococcus pyogenes aureus* has been followed by an endocarditis of ulcerative nature.

Micro-organisms are not present in any number in the blood. Direct examination of the blood gives as a rule negative results, and culture experiments have, except in a few rare instances, failed.

The Cardiac Lesions.

Two kinds of gross lesions are observed in connection with the diseased endocardium in malignant endocarditis. Of these, one is characterised by a loss of substance, and the formation of ragged and irregular ulcers; the other is distinguished by the growth of large and friable vegetations, that may attain a remarkable size. The two processes go on in nearly all cases side by side, though not as a rule in equal degree; and according to the predominance of loss of substance or vegetations the case is described as one of the *ulcerative* or of the *vegetative* variety of the disease.

Any part of the lining membrane of the heart may be involved in an infective inflammation. Lesions may be found on the endocardium that lines the cavities, or on that covering the valves. Both the left and the right side of the heart may be affected. Certain parts, however, are more apt to suffer than others; and the valvular structures, and those valves in particular that guard the mitral and aortic orifices are more often found diseased than other parts of the endocardium.

The incidence of inflammation is not, however, so closely dependent on the factor of local strain as in simple endocarditis. The infective form of inflammation is more prone to involve a larger area of the endocardium, and its extension to the mural endocardium or to the pulmonary and tricuspid valves is an event of relatively greater frequency than in non-infective endocarditis.

The ulcerative form.—The leading feature in this variety is a notable loss of substance. The destruction of tissue may be superficial or deep. It may be limited to the valves, or may involve other portions of the endocardium. In the earlier stages shallow ulcers are present, with ragged floors, and margins irregularly thickened by vegetations. If a valve segment be the site of ulceration, the structure may be weakened by thinning and soften-

ing of its tissue to such a degree that it yields to the blood-pressure, and a *valvular aneurism* is formed. The direction in which the valve yields depends on the relative strain on its two aspects: thus an aneurism of the mitral valve bulges into the auricle; aneurism of an aortic valve bulges into the ventricle. An aneurism formed in this manner may give way, or erosion of the substance of a valve may progress without aneurismal bulging until *perforation* of the valve segment is complete. After perforation has occurred the process of destruction may be continued, until ultimately the ragged remnant of the valve is detached, and carried away in the blood current.

In other instances the destruction of tissue commences at the periphery of the valve, and spreads thence towards the line of attachment, until the greater part, or even the whole, of the segment is destroyed.

When the endocardial lining of the cavities is affected, ulceration may advance until the muscular tissue of the heart is laid bare, and even becomes involved in the destructive process. The wall of the heart may in this way be so seriously thinned that it is unable to withstand the pressure of the contained blood, and, as in the case of the valve, a localised aneurism forms or perforation occurs.

Loss of substance, although so marked a feature of this form of malignant endocarditis, must not be regarded as peculiar to it. Ulceration is commonly present in the vegetative form, though here not so conspicuous as are the vegetations. Erosion of the endocardium is also of common occurrence in non-malignant forms of chronic endocarditis. The endothelial layer of cells is frequently wanting over a patch of atheromatous degeneration. The atheromatous material may subsequently be discharged and leave an ulcer. But destruction of tissue never occurs in such striking degree as in malignant endocarditis, when the valve segments may be completely disorganised and destroyed, and there may be nothing but a narrow rim left to indicate the line of their previous attachment.

The vegetative form.—Of this variety of malignant endocarditis the notable feature, as is implied by the name, is the formation of vegetations on the affected endocardium. These may be small and sessile, presenting no points of difference from the vegetations of simple endocarditis. As in simple

endocarditis, they are disposed more abundantly on the valvular portion of the endocardium than elsewhere, and are found with greater frequency on the aortic and mitral valves than on those of the right side of the heart. They display, however, a greater tendency to affect the lining endocardium and the valves of the right side than is usually the case with simple endocarditis. Patches of vegetation are found with some frequency attached to the wall of the left auricle, and in connection with the chordæ tendineæ.

More characteristic of malignant endocarditis are larger vegetations, of exuberant growth and irregular shape. In their proximal part they often are composed of organised connective tissue, possessing firm attachment to the underlying thickened endocardium. The tough proximal portion may be drawn out to form a pedicle, at the end of which the bulk of the vegetation hangs loosely. The peripheral part is usually less tough, and may be quite soft and friable. It consists of fibrin in the meshes of which are entangled blood-corpuscles and leucocytes, and it contains clusters of micro-organisms irregularly disposed in its substance. The vegetations are in some instances rendered stiff and coral-like by the deposition of lime salts.

Luxuriant though the growth of vegetation may be, with careful search there is nearly always to be found associated with it some loss of substance. This varies in degree from cases in which a close examination has to be made before evidence of ulceration is detected, to those in which the ulcerative changes are so pronounced that they rival the vegetative lesions in prominence, and there is doubt as to whether the case should be ascribed to the vegetative or to the ulcerative group.

A third or *suppurative* variety of malignant endocarditis is sometimes described, in which small collections of pus are found in the deeper tissues of the heart, either in the substance of the valves or in the muscular tissue. The abscesses, minute to begin with, increase in size, and ultimately burst through the endocardium into the cavity of the heart, leaving either superficial ulcers or more deeply-seated cavities, any one of which forms a point of weak resistance, and may eventually lead either to aneurismal dilatation or to rupture of the heart.

It is misleading to speak of this condition as a

variety of malignant "endocarditis." The abscesses are in most cases located in the substance of the heart wall, and have primarily no connection with the lining membrane. It is only secondarily that the endocardium becomes implicated.

The mode of origin of the abscesses is obscure. They are probably of metastatic origin, due to some pre-existing infective lesion elsewhere. The source of infection may indeed be an infective endocarditis, and in such an event the abscesses in the heart substance are analogous in their origin to the secondary infective processes that so commonly arise in other organs in the course of malignant endocarditis. They are, in short, due to an infective embolism of minute branches of the coronary arteries; and, rather than forming the starting-point of disease in a previously healthy endocardium, they are themselves the result of an antecedent endocarditis.

Embolism.

It will be readily understood that with a diseased condition of the endocardium such as has been described above, there is associated special risk of detachment of portions of the vegetative deposits, and their lodgment as emboli in distant organs. Embolism is, in fact, so common an occurrence in malignant endocarditis as to constitute a most important clinical feature, and one that is of the greatest service in differentiating between this and other forms of disease that may bear clinical resemblance to it.

The spleen and the kidneys are the organs in which emboli are most frequently arrested; less frequently in the brain, skin, intestines, retina, and other parts. Embolism of the lungs is prone to occur when the valves of the right side of the heart are involved.

The result of such vascular occlusion is the formation of an infarct, varying in size according to the area of tissue dependent for its blood-supply on the affected vessel. The area of infarction may pass through the familiar stages of hyperæmia and cicatrization, until ultimately a contracted conical mass of dense fibrous tissue is all that remains to indicate its position. The outcome is, however, often less fortunate. The emboli of malignant endocarditis usually contain infective micro-organisms, often of the pyogenic group. It is accordingly common for secondary centres of infec-

tion to arise wherever such emboli may be arrested, and the resulting infarcts frequently undergo softening, break down, and become the seat of abscesses.

Two points deserve special notice in the study of embolism in relation to malignant endocarditis. The first is an apparent caprice in the distribution of the infarcts. They may be present in large numbers in one organ while sparsely distributed elsewhere, or they may be limited to a single organ. In a case of malignant endocarditis infarcts were present in abundance in both kidneys, varying in size, but mostly small, pale in colour, and evidently of some duration, though not yet shrunken. No infarct was found in any other part of the body, although careful search was made. It is difficult to perceive the significance of so limited a distribution. There was in the state of the heart abundant cause for embolism.

The other point is that, notwithstanding the infective nature of the primary endocarditis, it is by no means an invariable rule that the secondary embolic centres become the site of suppuration. The explanation of this is also difficult. Micro-organisms are more abundantly disposed in some parts of the vegetations than in others; and it may be that of the detached fragments some contain no organisms, and are therefore non-infective. Or it may depend on the state of the tissues and their constituent cells, and their power to successfully resist and dispose of invading micro-organisms at an early stage, short of suppuration.

The blocking of an artery by infective clot is sometimes followed by a softening of its walls and the formation of an aneurism. In a case of malignant endocarditis in which there were many infarcts in the kidneys and spleen, a small rounded bulging of one renal artery was discovered, largely filled with clot, and projecting into the pelvis of the kidney. A similar but larger aneurism, also containing blood-clot, was found in the course of the superior mesenteric artery. Further, there was a fusiform dilatation of the basilar artery, which had ruptured, and given rise to an extensive hæmorrhage at the base of the brain. It appears from this that dilatation is apt to take place when the affected vessel is not running in the parenchyma of an organ, and therefore lacks the support of firm surrounding tissue.

(To be continued.)

ON CASES OF
**CHRONIC CATARRH OF THE MIDDLE EAR
CONNECTED WITH ATROPHIC
RHINITIS AND ATROPHIC PHARYNGITIS.**

BY

FREDERICK TRESILIAN, M.D., M.R.C.P. Edin.

IT will often be noticed that a certain number of patients suffering from ozæna are also the subjects of bilateral deafness. This is chiefly so in cases in which the original nasal disease has been neglected, and has either received no treatment at all for years, or has been very imperfectly treated, and therefore it is mostly in cases of advanced atrophic lesions of both nose and naso-pharynx that the disease of the ears occurs. This will be substantiated by the statement of the patients, to wit, that the nasal obstruction had been present for a long time before they began to notice the deafness, and that the onset of the deafness was very gradual, and proceeded *pari passu* with the progress of the ozæna. I believe that in these cases the condition is one of atrophy of the lining membrane of the Eustachian tubes, which may reach to that of the tympanum, and which leads to adhesive changes and secretion in both tube and tympanum, with, as a result, a form of obstructive deafness.

In many of these cases the appearance of the membrane is characteristic. It has lost most of its lustre, and, what is more, most of its natural mobility, as proved by the use of Siegle's speculum. The cone of light is diminished, and even lost in many cases, and the membrane looks dull and thickened. Thickening of the external layer of the membrana tympani, such as frequently occurs in cases of chronic external otitis, especially of the desquamative form, does not generally affect the mobility of the membrana tympani, but a thickening of the mucous or tympanic layer mostly does, presumably by affecting the mobility of the chain of ossicles.

In several cases also I have found diminished bone conduction, showing that the process is capable of affecting the cochlea to a very appreciable degree. The effects of ozæna of any long standing in causing wasting of the turbinated bones is well known, and it can be readily understood, if such a process goes on in the tympanum, that a somewhat similar osseous destruction may occur, with this

difference, that the destruction to a complete extent of the nasal spongy bones carries with it no marked symptoms or effects, but that a slight injury to the cochlea is capable of having a very marked influence on the range of hearing power.

As I said before, I believe from my own limited number of observations that it is only in old-standing and chiefly neglected cases of naso-pharyngeal ozæna that the tube and tympanum become affected by the extension of the ozænic process. I say neglected, because I have often seen advanced cases of ozæna in which, though the wasting of both bones and mucosa has been considerable, yet the patients keep the parts healthy and clean and free from crusts, and in such cases I have rarely known deafness to occur.

This is a subject which I do not recollect to have seen mentioned in any English text-book as a cause of Eustachian obstruction, yet it is one frequently met with clinically, and one which should always be remembered, because in some cases of ozæna the disease commences at the back of the nose, close to the choanæ, and in such cases the naso-pharynx and tubes may become affected without any evidence of much disease, at the time, in the anterior parts of the nose. Of course I do not wish to maintain that in all cases the atrophic mischief extends the whole length of the tubes, but that it often does I am well convinced, as that also in others it extends for a sufficient distance to cause a complete obstruction of the tubes; and in cases of this kind the tympanic affection may not be one of atrophy of the mucosa with secretion, but may be a true chronic catarrh of the tympanum such as is frequently found in old-standing cases of Eustachian obstruction, with adhesions and fixation of the stapes. In many cases there is also, either from the effect on the cochlea of the stapedial ankylosis, or a resulting extension of the lesion to the membranous canal, a certain amount of nerve-deafness resulting. In many cases I have ascertained the difficulty again and again of inflating the tympanum, either by the use of the air douche or catheter.

In most of such cases the rhinoscopic image is one of crusts, and when these have been cleared away the orifices of the tubes will present in many cases that peculiar stippled velvety appearance seen in the nose in commencing cases of atrophy. The chief therapeutical indication is for the use of

both post-nasal douche and post-nasal spray, the latter with a good strong bellows to try and force some of the fluid up the tubes; and after such cleansing I frequently apply with a probe some of Mandl's solution of iodine, but the difficulty of getting improvement is, in many cases, almost superhuman. I believe, however, in those cases in which the Eustachian tubes have been but slightly affected, that if the patient will, with the frequent help of his doctor, use the spray and douche regularly and vigorously (recollecting the tenaciousness of atrophic crusts) the deafness can in many cases be kept from progressing, which is certainly its natural tendency, once it does occur. It is well worth the patient's while to spend both time and labour in efforts to control it, for it is, on the whole, a much more serious matter for her or him than the loss of the turbinals or nasal mucosa.

VAGINAL versus ABDOMINAL SECTION FOR PUS IN THE PELVIS.

THE treatment of pus in the pelvis has passed through several transitional periods. The old unsatisfactory vaginal puncture gave place to the abdominal era inaugurated by Tait, and practised by his followers. The removal of pyosalpinx through the abdomen was the innovation which, under the ceaseless scrutiny of the statistician, made the field of abdominal surgery the "cloth of gold" of surgical battle-grounds. Then total castration through the vagina for double pus disease by the French school, and through the abdomen by the American school, engrossed the energies of gynæcological operators. These methods have reluctantly given place to modern vaginal section. I say modern advisedly, because it has an essential distinction from the old blind vaginal puncture, with the incomplete evacuation of perhaps one compartment of a multiple abscess, when it is contrasted with the free vaginal incision, careful exploration, and thorough evacuation of all pus pockets.

In its present application vaginal section constitutes the most recent acquisition to pelvic surgery, and it bids fair to revolutionise the results in pus disease. It should also be a subject of

gratulation that it is a distinctly American procedure. The assertion that the vaginal method is practised by men who are not expert as abdominal operators is incorrect. On the contrary, its employment in the pelvic inflammatory conditions has been evolved by men trained and thoroughly competent in the other operation. Peculiarly enough, the men who deprecate vaginal section as a blind procedure are the very men who ignore the advantages of the Trendelenburg position in abdominal work. Surely the fingers skilled in the enucleation of pus tubes through a small incision, unaided by the eye, can work equally well in similar manoeuvres *per vaginam*.

We should never forget, however, that the pathologic interpretation of pelvic inflammatory processes, now remedied in part by vaginal section, has reached its present wonderful perfection by those fearless and intrepid abdominal surgeons, who rescued the pathology of pelvic inflammation from the myths of antiquity.

In the present inquiry our motive should be, not to champion the one or the other method to the exclusion of the other, but rather to accentuate the relative worth of the would-be rival methods, and to determine, if possible, the positive indications and comparative merits of each. Unquestionably, the abdominal route affords facilities for visual inspection wholly wanting in the lower approach. The entire field of operation is kept under surveillance, and the attack on certain portions of the morbid masses can be made with entire confidence as to the safety of visceral integrity. Not so with the pus accumulations. If they are multiple, rupture and peritoneal soiling is inevitable, and that very circumstance is the supreme disadvantage of abdominal incision. While we have often seen the pelvis deluged with pus and no untoward symptom supervene, we have also seen patients rapidly perish within twelve hours from fulminant sepsis, the result of peritoneal contamination. Without doubt a large proportion of old pelvic abscesses contain so-called spent pus that can be spilled in the peritoneal cavity with impunity. On the other hand, there is that distressingly large class of cases that with singular and classical unanimity succumb on the critical third day to overwhelming sepsis.

There is no certain way of distinguishing these cases clinically, and hence all should be regarded

as virulent. This is a constant and irremediable menace. I have reported at another time a series of collected cases performed in the last year in five metropolitan hospitals in New York and Baltimore, with a mortality of 18.5 per cent. What must it be in the "unheard-from precincts" and in the hands of the great unwashed? This is no reflection on the reported results of many excellent surgeons who do laparotomy with mortalities of 2 and 3 per cent. I insist that this mortality does not include consecutive sections for pus, nor has it ever done so.

Abdominal surgeons have developed and perfected a most exquisite aseptic technique in detail and *ensemble*. They penetrate the abdominal wall in less than a minute with lightning dispatch. They enucleate with dexterity and assured safety to bowel and bladder. Manipulation is reduced to a minimum. Irrigation is deprived of irritation by physiological salt solution. The technique of glass drainage was perfected to such a degree that we were loath to exchange it for the easier and more efficacious vaginal drainage. Methods for homologous approximation of the abdominal wound have been devised that cause it to heal with beauty and surety, and with an inconsiderable number of subsequent herniæ. They accomplish all this with brilliant and sovereign celerity, and yet abdominal section as a routine practice for pus in the pelvis must inevitably fall into desuetude. Of course there remain many conditions where the abdominal route offers the best means of approach, notably tubercular inflammation of the ovaries and tubes. The removal of a small unilateral pus tube out of the true pelvis, or attached to the anterior parietes, is much easier through the upper incision; yet Polk and others advocate and practise anterior colpotomy for this condition.

The alleged limitations and difficulties of vaginal section are exaggerated. The procedure is comparatively in its infancy. Continued application will broaden and specify the limits of its utility, and increasing experience will augment our manipulative skill and perfect our operative technique.

In addition to the indications and supremacy of vaginal section for evacuating and draining pus in the pelvis, presently to be narrated, its most signal advantages have been exhibited in exploration of the pelvis for adherent adnexæ and small intra-pelvic tumours. With the exploring finger in

Douglas's space an accurate diagnosis of retro-uterine tumours, inflammatory and adnexial, can be easily made, and surgical measures immediately instituted for their relief. In this connection I will refer to the practicability of inspection of the pelvic contents through the vagina with the patient in the dorso-Trendelenburg posture (Pryor). This is readily accomplished by retracting the posterior wall and the opening in the fornix by the long retractor of Pean, and lifting the uterus upward and forward under the symphysis by the anterior trowel retractor. The intestines gravitate towards the diaphragm, and are further isolated by gauze pads. The appendages, if not adherent, or having been freed, gravitate into the exposed area, where any appropriate conservative procedure can be applied under guidance of the eye. I have also seen the appendix through the vagina, and the possibility of treating pelvic abscess of appendicular origin through the vagina has been proposed.

It is unnecessary to suggest the ease with which pus is reached through the vagina. It is the natural approach and logical drainage avenue of the pelvis and its contents. The natural history of pelvic pus accumulations is to become walled off above from the abdominal cavity. Opening and clearing out of these accumulations is virtually extra-peritoneal. It may then be classed in the category of minor surgery, but it gives major results. There is absolutely no shock. Patients thus treated give no more solicitude than a plastic case, and convalesce as smoothly as from a curetting. The entire absence of risk warrants us in urging a patient to have it done. And above all, patients so approached will give their ready consent. This is a very practical phase, and we cannot ignore the prejudice and possible refusal of patients, especially in private practice, to submit to more formidable operations.

We can change methods, but we cannot change the patient. Apart from these theoretical and general considerations is their practical employment. The application of methods to individual cases should be the determining factor here as elsewhere in surgery. We are too prone to make cases fit methods. In patients ill from prolonged sepsis, damaged kidneys demand short anæsthesia. Anæmia and asthenia preclude complete surgery, and simple vaginal section with drainage is elevated to the dignity of a life-saving procedure.

I would enumerate the special indications for vaginal section, aside from explorative purposes, in the three following classes of cases:

1. Early cases of acute suppurating salpingitis.
2. Incipient post-puerperal peritonitis.
3. Large pyosalpinx and true pelvic abscess.

In the first class will be found the cases from recent gonorrhœa and from septic abortion. As illustrative of the first type, I will mention the case of a girl nineteen years of age, who came to my clinic last summer with a fluctuating tender mass in the left side. She had had gonorrhœa a month, and presented herself with considerable pain and afternoon temperature. I curetted her in a hotel and made a posterior section. Upon incising the peritoneum the usual small quantity of free serum escaped. I found the tube fluctuating and tense. The right side was absolutely clear. I deliberately punctured the distended tube with scissors and withdrew them opened. A quantity of clear serum gushed forth, followed at the last by a minute quantity of pus and blood, that could be easily seen as it trickled over the blade of the depressor. The cavity was irrigated with saline solution, and packed lightly with iodoform gauze. The peritoneal opening was occluded with a small roll of the same material, which just entered it and filled the vagina. All gauze was removed on the third day. The peritoneal cavity had been entirely closed by lymph coagulum above the occlusive dressing. The sac cavity was re-irrigated and packed every second day. On the seventh day her temperature and pulse rose for the first time, and examination revealed a tender mass on the right side. On the eighth day I made another section above and to the right of the previous one, and found a "hydrosalpinx," in the descriptive rather than the pathological sense, which was in every way similar to the other one. I believe those serous effusions in the Fallopian tubes were the preceding pathological conditions to pyosalpinx.

If this be true, and is the embryonal history of suppurating salpingitis in early gonorrhœa and other inflammatory processes, the prophylactic value of vaginal section will be the greatest boon yet given to infected woman.

In incipient post-puerperal peritonitis Henrotin has taught us a simple lesson of pregnant truth. Associated with clearing and disinfection of the

septic uterus, vaginal section with drainage anticipates pelvic peritonitis and adhesions following puerperal infection. In these cases, at autopsies, I have seen literally puddles of pus in the cul-de-sac. The extension of the septic process and pus production was so rapid that nature had not time to encapsulate it. In this and in the ordinary adhesion cases of puerperal suppurative peritonitis it would be rash in the extreme to incur the dangers of supra-pubic section, where the simpler, more rational vaginal evacuation with uterine disinfection and drainage has everything in its favour.

Opening of large pelvic abscesses *per vaginam* needs no espousal of mine. It is nature's safest method, and was the practice of our elder criterions. I have seen the venerable Emmet evacuate large abscesses, and drain them by a permanent tube fixed into the vaginal incision by silver sutures. He told me he had done it in selected cases for over thirty years. It was then, as now, the operation of choice. While it must be regarded in most old cases as temporary, and undertaken for the relief of immediately dangerous symptoms, there still are many permanent cures. A case has been reported of incision of an ovarian abscess with subsequent pregnancy, the other ovary having been previously removed. There are doubtless many similar cases at least of restored functional activity in an ovary previously the seat of suppuration. Such reflections should make us chary of ruthlessly condemning appendages, especially ovarian abscess. "No organ, whose function can be maintained, should be sacrificed."

Should simple pus-letting not effect a cure, subsequent operation for removal of the relics of previous ravages can be done at another session without the dangers incurred in the presence of pus. This is the chorus of our contention.

In old recurring puriform disease, where both annexæ are so hopelessly destroyed as to demand extirpation, I believe the uterus should also be removed. In such cases the condition of the patient forms the only contra-indication for complete ablation; not simply because it is a functionless organ and can be removed with low mortality, but because it too is diseased, and if left will continue to produce pain and prolong the disturbances of the artificial menopause; it may

still be the seat of hæmorrhagic discharge, may be infected or re-infected with gonorrhœa, harbour tubercular bacilli and other germs, and incubate cancer cells. In destructive bilateral suppurative disease of the appendages the uterus is enlarged by plastic exudation, may be infiltrated with pus or permeated with latent gonococci. The adhesions binding it in vicious malpositions are intensified, after the removal of the purulent extension processes, by re-adhesion of hollow viscera to denuded areas on the uterine wall.

Whenever the uterus is diseased by pyogenic infection beginning in its own cavity and extending and destroying the function and integrity of its appendages, it should be removed. The sub-pubic operation is preferable to the supra-pubic for the same reasons that vaginal section is preferable to abdominal section for pus in the pelvis. Moreover, it has been demonstrated that "whatever can be enucleated through the abdominal wall can also be removed through the vagina, and whatever it is impossible to enucleate through the vagina cannot be removed by the abdominal method, except at the price of procedures incomparably more grave and more laborious."

The field of vaginal section is to prevent supuration in early cases, to anticipate it in puerperal cases, and to save life in desperate pus cases. It is simple, surgical, and safe. Its application to the pelvic inflammatory diseases of women and to pus in the pelvis is one of the greatest surgical triumphs of the age.—William D. Haggard, jun., *American Practitioner and News*, Nov. 28, 1896.

NOTES.

The Heart in Typhoid Fever.—According to Dr. F. Plicque, the cardiac complications constitute, in typhoid fever, one of the principal causes of death. Some form of heart lesion is found in more than two thirds of all autopsies. Dry pericarditis is rare; also pericarditis with effusion is rare. Endocarditis is more common. But a myocarditis, from an infection by the Eberth bacillus, is very common, and this is the characteristic heart lesion in typhoid fever.

It is especially in the exhausted, in the obese, in the alcoholics, and in individuals prone to heart lesions that this typical myocarditis is found.

The hyperthermic plays an important causative

role, and, consequently, the cold bath treatment has a prophylactic value. But, on the contrary, a too rigorous application of the Brand treatment in nervous subjects will induce cardiac complications.

The myocarditis may be due to a degeneration of the muscle fibres; or to paresis of the trophic nerves; or it may be infectious in nature.

The stethoscopic signs, which can call attention to the cardiac complications, are variable. In the grave forms, it is often at the beginning of the second week that one can notice a great attenuation of the first sound. The apex bruit becomes weak and diffused. The attenuation of the first sound may progress until it disappears. The confusion of the second sound is more rare, but a reduplication of the second sound is frequent. Coincident with the attenuation of the first sound, Hayem has noted the substitution of the præcordial impulse by a vague undulation.

Murmurs most always precede, for a variable space of time, the confusion of the sounds. The murmurs are usually soft, variable, systolic, with the maximum intensity a little above the apex.

The disturbances of the cardiac rhythm are of more importance than the murmurs. The so-called "fatal" rhythm or pulsations that resemble the tic-tac of a watch are very important. The intermittences may be regular, as in some cases every fourth pulsation is lost.

The pulse, which in typhoid fever is relatively slow, suddenly increases in rate when a cardiac complication (especially myocarditis) occurs. The danger of cardiac failure is threatened when the pulse rate reaches 120 per minute. An accelerated pulse, with a relatively low temperature, is an indication of cardiac collapse. In place of a soft, full, and compressible pulse, it may become small and hard.

The physical signs and appearances are very important. Functional disturbances are not often noticed. There is rarely pain or palpitation.

If patient is pale, and if the lips and extremities are cold and slightly blue, the prognosis is not good, because these phenomena indicate progressive heart failure. The appearance of cedema, however light, is a very grave sign. A profuse cold sweat on the extremities is an unfavorable sign. Often the disturbance occasioned the patient by moving him in the bed, will cause extreme paleness and sweating. This instability

of the heart as shown by the pulse becoming small and irregular when the patient is shaken, is of very grave signification.

Typhoid fever victims are constantly in danger of syncope. Death may occur in the first collapse, or two or three attacks may occur before a fatal termination. When the syncope is prolonged the outcome is almost always fatal. These attacks of syncope may come late and after a period of apparent convalescence. They usually occur at the end of the third week, and they are more frequent in cases of moderate severity which are not so vigilantly watched as graver cases.

Death may ensue slowly from gradual heart failure. Hypostatic congestion and pulmonary cedema are often a herald of cardiac failure. Inter-cardiac coagulations can also occur when the circulation becomes slow, and become the origin of emboli in the arteries of the extremities, spleen, kidney, &c.

To prevent these grave heart complications, the best means is found in a rigid application of hydrotherapy, according to Brand. Some administer the cold baths every two hours at 16° C. To support the heart and to antagonise the shocks of the cold plunge it will be well to inject, subcutaneously, sparteine and ergotine. This treatment is the surest method to stimulate the vascular tone when it begins to lag.

In very nervous, impressionable subjects who have had previous heart troubles, the cold pack may be substituted for the bath. Towards the end of the fever, especially at the end of the third week, it is necessary to prohibit all sudden movements. It is absolutely essential to maintain the patient in a horizontal decubitus. Any sudden exertion or change of position may cause heart paralysis.

If pulmonary stasis (hypostatic congestion) occurs, the patient can be carefully rolled on one side at stated intervals.

The administration of nourishment in a scientific way does much to obviate cardiac complications. When there is evidence of general heart weakness and failure the hypodermic use of cardiac stimulants must be begun: ether, caffeine, sparteine, camphor.

Alcoholic frictions of the extremities and an envelopment of the limbs with hot applications are useful in a collapse.

In tachycardia an ice-bag placed over the præcordial region will have a good effect.

Indian Lancet, April 1st, 1897.

THE CLINICAL JOURNAL.

WEDNESDAY, MAY 12, 1897.

A NOTE FROM THE CLINIC

OF

DR. CROCKER, AT UNIVERSITY COLLEGE
HOSPITAL, MARCH 30th, 1897.

Leprosy.

CASE of A. E—, No. 278, 62 years of age, male. This patient tells us that three and a half years ago he was quite well; he was then on a voyage round Cape Horn, when he contracted what he called "scurvy," and had bleeding from the gums. Five years ago he was working in the East Indies. The man has been a seaman all his life, has been all over the world, and dates his illness from the "scurvy," and has never been well since.

The exact time when his face began to alter cannot be ascertained, but he thinks about a year ago his friends noticed it. The face now has a very characteristic expression. It is uniformly of a yellowish brown tint. The vertical and the horizontal lines of the forehead are much deepened, especially between the brows. The greater part of the eyebrows have fallen out, mostly on the right side. The eyelashes are completely gone, and the lids are distinctly yellowish, and slightly thickened. The prominence of the brow is not greatly exaggerated, and the thickening is not very marked when pinched up. The skin over the orbit in the lower portion is baggy, but from infiltration and not from œdema. The cheeks have sunk in the centre, but the naso-labial folds are notably exaggerated. The skin beyond the hollow forms a loose fold. The nose is much swollen and infiltrated; the patient has a thin beard and moustache, but has never had whiskers. The lines round the mouth are deepened from the infiltration; the lips, however, are only moderately thickened. The patient is evidently much emaciated, so that the face looks elongated. The lobes of the ear are longer and thicker than natural, the left one being nearly an inch long. The lobe of the ear is thickened to the touch, and is discoloured

like the rest, but does not present much alteration otherwise. The conjunctivæ are very slightly congested, but do not suggest any active disease. The tongue has a nodule on the left tip, and the surface is transversely furrowed, but it does not show any other infiltration. On the right side there is slight infiltration, and on the left over the nodule are some white linear cicatrices. The patient has lost all his upper teeth except two, and there are not many teeth in the lower jaw. The uvula is notably infiltrated, and forms a small cherry sized tumour. There is a very slight small nodule behind the right anterior pillar. The roof of the mouth is infiltrated, white, and at one point slightly ulcerated. There are also white lines traversing it, doubtless of a cicatricial character. The skin of the trunk in front is slightly discoloured, with a yellowish tint above the right breast, and to a less degree on the abdomen, but otherwise presents no notable alteration except thinning. On the neck, however, the brownish infiltration extends about halfway down, the sub-maxillary glands are notably, and the post-sternomastoid glands are slightly enlarged. Occipital glands cannot be felt. In the lower part of the neck the follicles are unduly prominent, looking as if the skin were crowded with milium. On the back, there is some yellowish discoloration about the shoulders, and a small lipoma in the left interscapular region, but otherwise there is nothing abnormal.

There is very marked discoloration on the whole extensor aspect of the upper limbs, which is most marked below the elbow and on the hands. There is no noticeable infiltration in the upper arms, but in the forearm it is distinctly perceptible when the skin is pinched up on the extensor aspect. The back of the hand and fingers is also notably infiltrated, but at the same time the skin is much wrinkled and quite inelastic, and here the brownish hue becomes slightly cyanotic towards the knuckles and fingers. There is a hæmorrhagic bulla on the radial side of the second finger, while on the left side the nails are not altered. Everywhere on the forearm and hand the smooth

satiny characteristic feeling is well marked. On the flexor aspect discoloration is much less marked on the forearm, and there is very little infiltration. The palmar surface is hard, dry, and powdery, with exaggerated natural lines, except on the finger tips, which are preternaturally smooth and slightly reddened. The patient has a sore on the right hand corresponding with the bulla on the left second finger, which he remembers began in a blister, the same as the one now present on the left side; in other respects the description for the right is same as for the left limb. The ulnar nerve can be distinctly felt at the elbow on the left side, but not so well on the right. Roughly speaking, there is anæsthesia over the whole of the back of the hand and the extensor surface of the forearm, and to some extent about halfway up the back of the arm, but this requires to be more carefully mapped out. There is nothing noticeable on the buttocks and backs or fronts of the thighs except some yellowish mottling, and the skin is dry, wrinkled, and powdery. On the lower half of the right leg there are two ulcers in front, one two and a quarter inches and the other one inch in diameter. The skin round them is red, but the edges do not look unhealthy, and except at the lower part of the larger ulcer the base is for the most part clean, with not unhealthy-looking granulations. On the right foot, the skin is infiltrated and puffy like the hand, but the purplish hue is less marked. On the left leg, there are two small ulcers half an inch in diameter, nearly symmetrical with those on the right, but rather lower down. There is a scar over the left knee and another above it, which he attributes to scurvy ulcers; there is a similar scar above the right elbow. The soles are dry and powdery-looking, but the skin is comparatively soft except over the right big toe, where there is a small callosity. Always look at the soles for perforating ulcers, as they are very common in this condition. There is some anæsthesia in the lower extremities, which has not yet been mapped out. There are greatly exaggerated knee-jerks, and clonus is well marked.

This is a case of tuberculated leprosy. There is the characteristic appearance of the face, the exaggeration of all the lines and folds, not from the actual deepening of those lines, but from the infiltration on both sides, and the so-called leonine appearance so well marked in him is due to that.

If you get rid of the infiltration the lines become proportionately shallow, and his appearance will improve correspondingly. It is not a very favorable case for treatment; the patient is emaciated, and looks cachectic generally. You see also he has a neuritis, which affects chiefly the peroneal and ulnar nerve trunks, but it may affect any of them.

Sometimes, as in this case, the median is involved as well, as may be inferred from the distribution of the anæsthesia. The bullæ on the fingers are, no doubt, a sequel to the neuritis, and are due chiefly to nutritional changes from the cutting off of the nerve supply. The nerves act as protectors to the skin and tissues. If you cut them off, slight injuries will produce lesions such as bullæ, which would not happen otherwise. Quite early in the disease it is common to have symptoms of peripheral neuritis, and then you may have an acute outbreak of bullæ through the direct result of the neuritic change. Here, in this case, they are from the late effects, and may occur from burns and injuries, and are of the same class as perforative ulcers, which are common in the foot, and do occur in the hand, in leprosy. These yellow discolorations are remains of former erythematous eruption. One of the early signs is an erythema of the skin somewhat analogous to the eruptions that we see in syphilis and other similar diseases, a symmetrical erythema with much more decided infiltration, and more persistent in its manifestation. Sooner or later the erythematous eruption fades away and leaves a yellowish discoloration with or without infiltration. Here the infiltration is clearing up, and we have only the staining. On the forearm, there is still left a considerable amount of infiltration. There is a soft satiny feeling of the skin at the extremities which may be noticeable in the face, sometimes associated with the greasy feeling, and sometimes actually with greasy seborrhœa. In other parts there is dryness with its powdery appearance, well marked on the soles and palms.

One is in the habit of dividing leprosy into cases affecting the skin and cases affecting the nerves, but there is no real difference between them. The brunt of the disease in one case falls on the skin, and in the other on the nerves. They are frequently mixed up together, for there is no hard and fast line, and so you may have a combination of symptoms. Many authors divide leprosy into three clinical types: the tuberculated type, corresponding

to the skin form ; the non-tuberculated, mainly or almost exclusively attacking the nerves, sometimes called the anæsthetic form ; and the mixed condition, when nerves and skin are simultaneously or almost simultaneously affected. This division is of some practical importance because it indicates to a considerable extent the course of the disease : the mixed is the most rapid, the skin or tubercular is the next, and the nerve form is, generally speaking, the slowest ; but both the tuberculated and the nerve form are often extremely slow, taking a great many years to produce the result which generally ensues sooner or later. Twenty years for the nerve form is nothing very unusual. That, however, largely depends upon whether it remains a nerve form throughout, or whether the skin becomes involved. Sooner or later you have, as a rule, the combination of nerve and skin. I am distinguishing now between what may be called the primary and secondary skin lesions, for in the nerve form we may have secondary lesions that have no great effect on the ultimate result. Under favorable circumstances the tuberculated form runs a course for seven years, while the mixed is from three to six years. There are exceptions, of course. I have had a gentleman with tuberculated leprosy under my care for nine years, who is now much better than when he first came under treatment.

The early symptoms of the skin form of this disease, are always interesting and sometimes misleading. They often start with marked febrile symptoms, often intermittent, and are supposed to have ague, and possibly do have it sometimes, preceded, followed, or accompanied by profuse sweating, vertigo, lassitude, listlessness, depression and drowsiness, and gastro-intestinal disturbance. Generally lassitude and a marked disinclination to exertion of all kinds is to be remarked in the tuberculated cases, and after a variable period of days, weeks, or months, the erythematous exanthem appears. Sometimes the eruption develops gradually, with scarcely noticeable general symptoms. If the nerves are involved, then you have shooting and darting pains, and these pains become sometimes of an excruciating character, and render the patient's life utterly miserable ; but when the nerves escape, the patients have practically little suffering, and the course is often so gradual that, with the exception of the disfigurement, it is

one of the diseases in which the patient suffers little. Actual suffering depends upon whether there is active neuritis or not ; where that is absent, beyond the lassitude, and the almost "don't care" sort of feeling that comes over them, there is little to complain of. The difficulty is to rouse these patients to take sufficient trouble to adopt and persist in the means to improvement that are open to them. The remedial measures are always slow in action, and require much co-operation on the part of the patient and attendants. There are two ways of anæsthesia being produced, from neuritis of the nerve trunk, such as in the ulnar, or from infiltration of the skin blunting the ends of the nerves, and possibly leading to changes in the ends of the nerves, partly from pressure, or from a low form of neuritis in the ends of the nerves themselves.

Our patient has some peripheral neuritis, as evidenced by his knee-jerks, and from the condition of the skin, but it is mostly due to the involvement of the nerve trunks. If the anæsthesia is only due to infiltration of the skin, you do not have that definite nerve distribution, and you do not find that the nerve trunks themselves are involved. If the mucous membranes are affected, as in the larynx, that modifies the course of the disease. If they have tubercle in the lung naturally that will shorten the course.

It is a disputed point whether there may not be actual phthisis due to this form of leprosy. The bacilli of both diseases are very closely identical in their size and appearance, and are identical in their staining characters. It is not unlikely that they set up similar lesions in the lungs, and it is a moot point whether there is a true phthisis of this kind independent from the ordinary tubercular phthisis, which is no doubt apt to occur in any chronic disease leading to a lowering of the general nutrition. It is quite possible that there may be on the one hand tubercular phthisis due to want of nutrition, and there may be a phthisis directly due to this leprosy.

The two chief remedies for this disease are, first, chaulmoogra oil, and the other we have found out lately here is intra-muscular injection of perchloride of mercury. Some of you have seen the effects of that on some cases, and I have seen it in others, and so I hope that this case will respond to treatment. There can be no doubt that chaul-

moogra oil is of use provided—and this is possible only in a small number—that the patient can take enough. It is a nauseous remedy. You have to begin with three minims three times a day, and gradually work up to large doses, such as 100 minims a day; if he can take that his prospects are very much improved, but if, on the other hand, he cannot get above twenty or thirty drops per diem, there is not much good in the treatment.

Lately I saw a gentleman from Peru, who had apparently perfectly recovered; there was nothing wrong with him, and I should not have known he had had leprosy except that there was a little anæsthesia in the hands and in one foot, for if the nerve fibres have been destroyed by the pressure of the leprosy material nothing can restore them, and you cannot get rid of the anæsthesia by any possibility. Although, therefore, the anæsthesia remained, this patient appeared to be perfectly well. He told me that by living in the mountains he had reached as high as 500 drops of chaulmoogra oil a day, and he immediately began to improve, while in the plains he could only take small doses. Sporadic cases of cure in the nerve form are not so rare, but there will be deformity left. At San Remo I was shown one of these cases of cure except as regards deformity. Perfect cure of fully-developed cases of the skin form has hitherto been regarded as almost impossible, but sometimes when there have been only a few nodules present apparent cure has resulted. The beneficial effects of perchloride of mercury injections have been so marked in cases that would formerly have been regarded as hopeless that one is tempted to hope that even some of these may recover; but time alone will show whether the marked amelioration observed is temporary or permanent. In any case it yields better and more rapid results than have hitherto been obtainable by other means.

A Powder for Ulcer of the Leg.—

Finely powdered chloride of sodium, 10 drs.

Powdered menthol 1 dr.

Mix thoroughly and use as a dusting powder after thoroughly washing the surface of the ulcer clean.

This is particularly useful in atonic slow ulcers of the leg, and aids very materially in maintaining antiseptis and producing healing.

Journal de Médecine de Paris, 1896.

ON SOME PAINFUL AFFECTIONS OF THE FEET.

By A. H. TUBBY, M.S.Lond., F.R.C.S.Eng.,

Assistant Surgeon to, and in charge of, the Orthopædic Department, Westminster Hospital; Surgeon to the National Orthopædic Hospital; Surgeon to Out-patients, Evelina Hospital for Sick Children.

PART III.

Metatarsal Neuralgia, or Morton's Disease.

THIS condition is a neuralgia chiefly situated at the anterior part of the foot, especially about the head of the fourth metatarsal bone. In most cases the pain is very acute, but in slighter cases it consists merely in a dull ache. It was first described in 1876 by Dr. T. G. Morton under the title of "A Peculiar Painful Affection of the Fourth Metatarso-phalangeal Articulation." Since then it has received notices from the following writers:—Dr. Bradford, of Boston, under the title of "Metatarsal Neuralgia, or Morton's affection of the Foot," and described in the 'Boston Medical and Surgical Journal,' 1891, vol. ii, p. 52; Dr. Virgil P. Gibney also wrote on the non-operative treatment of metatarsalgia in the 'American Journal of Nervous and Mental Diseases,' September, 1894, p. 589. Other writers have also noticed it, especially Dr. Goldthwait, of Boston, Dr. T. S. K. Morton in the 'Transactions of the Philadelphia Academy of Surgery,' 1893; and in this country Mr. Edmund Roughton in the 'Lancet,' March, 1880, and Mr. Robert Jones in the 'Liverpool Medico-Chirurgical Journal' for January, 1897. Mr. R. Jones gives a very full and exhaustive account of the affection, and has done much original work on the subject. He describes three degrees of metatarsal neuralgia. These degrees are useful from the symptomatic point of view, and more especially so as all the authors who have written on the subject agree that the immediate cause is pressure on one or more of the digital nerves at the heads of the metatarsal bones. According to Dr. T. G. Morton, the pain is localised at the interspace between the fourth and fifth metatarsal bones; but in four of the cases I have seen it has been present in the second or third interspaces, frequently

starting about the head of the third metatarsal bone.

With reference to the causation, there can be no doubt that the rheumatic or gouty diathesis plays an important share in the production of the disease, but its incidence is determined by a blow or a strain, or a fall in which the weight comes mainly on the front part of the foot. In other instances it supervenes after long standing and walking, especially in narrow boots, and occasionally the disease follows a long illness. In many cases some degree of flat-foot is present, but occasionally one finds it associated with the reverse condition, namely, the hollow or claw-foot. Its association with flat-foot is very interesting from an ætiological point of view, since there can be no doubt that with the ordinary condition of flat-foot there is associated a falling of the anterior transverse arch, in such a way that pressure is made upon the digital nerves, or upon the communication which exists between the digital nerves of the third and the fourth toe, as Mr. R. Jones has recently pointed out.

Symptoms.—Firstly, the attention of the surgeon is called to the patient's foot on account of the pain suffered; frequently it is intense and paroxysmal and renders movement impossible—such are the severe cases; nor is it confined to the foot, but starting about the head of the third or fourth metatarsal bones it is reflected up the limb. As a rule, no redness is present, but I have seen very considerable congestion in one case. In that instance, while at rest the patient would suffer little or no inconvenience, and would start on a walk without discomfort; shortly afterwards the pain commenced, and became worse on going into a warm room, the feet then felt hot and the patient was unable to move; his one desire was to remove the boot and to hold the front part of the foot firmly. This description of the pain is very characteristic, and in almost all cases the patient will volunteer the statement that there is nothing gives so much relief to the pain as removing the boot and holding the instep. On two occasions during a severe attack of pain I have found redness present about the front part of the foot, especially in the third and fourth interspaces. In the less severe cases the pain is not always of this acute nature, but is occasionally of a chronic character.

Secondly, deep tenderness is present about the heads of the third and fourth metatarsal bones.

Mr. Robert Jones has pointed out that in almost all cases pain may be elicited by firmly holding the head of the fourth metatarsal bone between the finger and thumb. In moderately severe cases it requires somewhat continued pressure to do this, but in bad cases the slightest pressure causes exquisite pain.

Thirdly, the affected foot is broader across the heads of the metatarsal bones than is normal, and this observation goes with the condition of flat-foot, which is frequently found in these cases; indeed, the anterior transverse metatarsal arch has fallen.

Fourthly, on examining the sole, a large corn may be seen over the heads of either the second, third, or fourth metatarsal bones, one of which is felt to be prominent in this situation. This prominence of the head of one of the metatarsal bones, taken in conjunction with the character and the starting point of the pain, are diagnostic of the disease. Oftentimes the corn is situated over the fourth metatarsal bone, and this is one of the points of the greatest pressure on the soles of the feet.

Fifthly, Goldthwait has observed a peculiar twist of the foot, the portion in front of the tarso-metatarsal articulation being twisted inwards, so



Fig. 18.—Tracing of the foot from a case of metatarsalgia.

that the base of the fifth metatarsal bone is exposed to the pressure of the boot, and the patient complains of constant pain in that spot. In fact, in some cases—and these are early instances—the patient seeks relief from this alone.

Sixthly, a tracing of the foot is typical. There is a bulging instead of a re-entering angle behind the ball of the great toe (Fig. 18). In illustration of these points I may quote one or two cases.

A gentleman æt. 32 consulted me for paroxysmal pain in the front part of the right foot, which became so severe at times as to entirely prevent him from moving about. He played at cricket a great deal, and had frequently been struck with a ball on the dorsum of the foot. He was wearing rather narrow-pointed boots, and no doubt these contributed to the perpetuation of the pain. The latter was always worse in the evening, and occasionally became agonising in a warm room; it was accompanied by considerable redness and extreme tenderness in the first interspace. He could only obtain temporary relief by removing the boot, which he was obliged to do at all hazards.

On examination it was noticed that the arch of the foot had fallen, the base of the fifth metatarsal bone was prominent, the anterior part of the foot was twisted inwards, and there was depression with enlargement of the head of the second metatarsal bone. Relief was obtained by rest, and by the use of boots of the following description: made tight across the instep and very broad across the toes, together with a high instep. He was also advised to soak his feet in hot water containing a drachm of bicarbonate of soda to the pint. Citrate of potash was given internally. After some weeks the pain lessened and disappeared.

Another case was that of a gentleman æt. 25, who consulted me in July, 1895, with reference to pain and difficulty in walking. He could only hobble on account of the pain, and had tried all sorts of boots. The history of gout was well marked in the family. Pain was complained of in both feet, about the head of the third metatarsal bones, and over the base of the fifth metatarsals. In the soles of both feet the head of the third metatarsal was very prominent, with a large corn on it, and on the right foot small ones were present over the heads of the second and fourth. The arch of the foot was much increased, and the toes of both feet were hyper-extended. At times he had acute attacks of pain lasting on and off for a fortnight, and he was completely laid up. The inward twist of the foot was well marked, and so extreme was the displacement of the head of the third metatarsal bone on the right side

that I advised its removal. As he objected to this, and was anxious to try other treatment, I advised bathing in hot water every night, and the wearing of boots closely fitting over the instep, and very broad in the tread. In November, 1895, I heard that a considerable improvement had occurred.

Another case was that of a gentleman æt. 35, in whom the pain was so severe that he was unable to walk more than three or four hundred yards at a time. In his case the pain became worse shortly after setting out for his office in the morning, and when I examined him I found that there was very considerable tenderness over the second and fourth metatarsal bones in both feet, but more marked over the second. He preferred to seek relief by operation; and as the pain was worse about the second metatarsal bones, I removed the heads of these bones, and in doing so I noticed that the digital nerves were dark red and much swollen. There was some delay in the healing up, but ultimately the patient was benefited by the operation.

Pathology of this affection.—The explanation given by Dr. T. G. Morton is as follows:—The heads of the first three metatarsal bones are nearly on a line with, and less moveable than the remaining ones; the head of the fourth is a quarter of an inch behind that of the third, while that of the fifth is nearly half an inch behind the head of the fourth; and between the heads of the fourth and fifth, branches of the external plantar nerve pass, while the anterior extremity of the fifth metatarsal and to a less degree of the fourth are very mobile. When the transverse arch is compressed, the head of the fifth metatarsal bone and its proximal phalanx come directly into contact with the head and neck of the fourth metatarsal, and consequently the nerves are compressed. While this anatomical explanation suffices in the case of the fourth and fifth metatarsal bones, it does not explain the instances of metatarsalgia beginning between the second and third and third and fourth bones. It has, however, been stated that most frequently the pain is about the head of the fourth metatarsal bone, and Robert Jones, of Liverpool, has brought forward an explanation which much more correctly fits in with the facts. He has shown that the communication between the fourth branch of the internal plantar nerve and of the

external plantar nerve takes place near the head of the fourth metatarsal bone, and Robert Jones is of opinion that clinical observations accord much better with the theory of treading upon rather than pinching of the nerve; and he advances three anatomical facts, namely, the proximity of the painful area to the communicating fourth branch of the superficial division of the internal plantar, the collapse of the anterior arch, and in most cases the fact that the bulk of the superincumbent body-weight in walking on the toes is borne on the first and fourth joints. He does not agree with Gibney that by grasping the bases of the metatarsal bones, the heads are separated, but he finds that the pain is relieved by grasping the foot around the head of the metatarsal bones, thus lifting away the heads of the bones from the irritated nerve.

Diagnosis.—This must be made from flat-foot chiefly by reason of the pain. It has already been stated that in many cases of Morton's disease, flat-foot of a minor degree is present, but it is rarely so marked as to explain the acute and agonising pain of metatarsalgia. Cases may be considered to partake more of Morton's disease than of flat-foot when the pain begins about the heads of the metatarsal bones, and is of the paroxysmal nature already alluded to. In some instances, however, not a trace of flat-foot exists, but the arch of the foot is exaggerated, so that in this instance no confusion ought to arise. Again, in Morton's disease the foot is usually of a healthy colour and appearance, whereas in painful flat-foot it is blue and congested.

Prognosis should be guarded in all cases, for even with complete rest the acute pain diminishes slowly for a few days, and for weeks afterwards exacerbations may take place when the patient walks. These are due to the accompanying neuritis. The effects of treatment are displayed slowly, and the patient should therefore be warned that the trouble is likely to be a tedious one, except when it is operated upon.

Treatment.—In all instances evidences of rheumatism, rheumatoid arthritis, and gout should be sought for and treated with the usual remedies. The acute attacks of pain can be relieved by removing the boot and soaking the foot in hot water. The application of the oleates of morphia and atropine will temporarily relieve the pain. In some cases, relief for a time may be obtained

by grasping the instep and sole, or by tightly compressing the bases of the metatarsal bones with a flannel bandage. In moderate cases the first thing is complete rest to the foot for two or three weeks, then the patient may be allowed to walk; but no boot should be worn for a time, merely a canvas or rubber shoe with a bandage round the proximal ends of the metatarsal bones. When all pain has subsided the following description of boot should be worn, namely, one with a high instep and valgus pad, if flat-foot is present, with the heel coming well forward beneath the instep, and of moderate height. The boot should be also made to fit closely over the instep, and must be broad in the tread so as to give plenty of room for the heads of the metatarsal bones, and the soles must be thick. An excellent arrangement for moderately severe cases is suggested by Mr. Robert Jones in the 'Liverpool Medico-Chirurgical Journal' of January, 1897. He recommends that the boot should be one quarter of an inch thicker behind the heads of the metatarsal bones than elsewhere, and in severe cases that there should be a thick bar of leather placed about half an inch behind the heads of the metatarsal bones. In cases of great severity, and in those which refuse to yield to treatment of this description, an operation must be carried out. The best form of operation is undoubtedly the removal of the head of the metatarsal bone around which the pain is greatest. As before stated, in the majority of cases this will be found to be the fourth. Some recommend excision of the joint, and others merely neurotomy, but this latter procedure is of little avail. It is sufficient in nearly all cases to exsect the head of the bone. Over the head of the metatarsal bone a longitudinal incision is made on the dorsal aspect. The extensor tendon is divided and half an inch of it taken away, and the head is removed with a pair of bone forceps. The patient rests in bed for a week or ten days, and does not walk on the feet for about two to three weeks after the operation, when the wound will have become consolidated. The success of this operation is very great indeed, and the relief is little short of marvellous.

(To be continued.)

ON THE ÆTIOLOGY, PATHOLOGY, AND CLINICAL VARIETIES OF MALIGNANT ENDOCARDITIS.

BY

ARTHUR G. PHEAR, M.D., M.R.C.P.,

Assistant Physician and Pathologist to the Metropolitan
Hospital.

IN TWO PARTS.

PART II.

Symptoms and Clinical Course.

THERE is great difficulty in giving an adequate clinical account of a disease which presents itself clinically under so varying an aspect as malignant endocarditis. The study of a number of cases shows wide divergence in clinical detail; individual symptoms display marked variation in character; those that are prominent in one case may be absent in another.

Nevertheless it is found that the clinical manifestations of malignant endocarditis fall, almost naturally, into three groups, and a clear idea of the disease may best be gained by making a separate study of each group.

1. The first group includes general symptoms, mostly of *pyæmic* nature, and similar both in origin and character to those met with in other forms of pyæmia.

2. In the second group, which from the clinical point of view is often the least conspicuous of the three, are included the symptoms and signs of *cardiac* origin, due to defects in the working of the valves, and resulting inefficiency in the performance of the cardiac function.

3. Among the symptoms of the third group are those of *embolism*, due to the detachment of portions of vegetation or of thrombi formed in connection with the diseased endocardium, and their arrest as emboli in distant parts of the body.

1. Among the general symptoms of malignant endocarditis, *fever* occupies an all-important place. In the absence of fever the diagnosis of malignant endocarditis cannot reasonably be made.

Although a symptom of constant occurrence, it is, however, remarkably variable in its character. So diverse are its features that certain clinical types

of the disease have been differentiated, according to the aspect of the temperature chart. Thus a *typhoid* type of malignant endocarditis is described, in which the daily range of temperature is between 1.5° and 2.5° , similar to that of typhoid fever at the height of the disease, though wanting in the precise regularity that may be observed in typhoid fever, and of course lacking the characteristic rise and fall that is seen at the commencement and at the decline of typhoid fever. In the *pyæmic* type of malignant endocarditis the daily variation is much greater. Each day the temperature may fall to the normal line or below it, though rising to 103° or 104° or higher. When fever of this type is present, rigors are of frequent occurrence with the rise of temperature, and profuse sweatings accompany its fall. The case presents all the features of hectic; the patient loses flesh and speedily becomes anæmic, and the disease makes rapid progress. More remarkable are those rare cases of malignant endocarditis in which the temperature simulates that of *malaria*. This form of the disease is characterised by pyrexial attacks, alternating with intervals of a day or two days, during which the patient is free from fever. The rise of temperature is accompanied by a shivering fit or a definite rigor, and the fall by copious perspiration. There may be considerable superficial resemblance to an attack of ague; but it is very uncommon for the pyrexia to be so strictly periodic as to lead to real risk of confusion; and in cases of difficulty valuable clues to the nature of the malady are afforded by the inefficacy of quinine in warding off or controlling the pyrexial attacks, by the failure to detect the malarial parasite in the blood, and in particular by the recurrence of embolism with other direct evidence of cardiac disease.

In many cases of malignant endocarditis, however, it is notable that the fever bears no resemblance to that of typhoid fever, nor to that of malaria. Nor does the temperature chart display the regular and wide daily variation of pyæmia; but the fever runs an *irregular* course, conforming to no type, and characteristic in no respect save that it is long-continued. Even when, for a time, the temperature is fairly uniform in its degree and daily variation, it may, as the disease progresses, lose character and become irregular.

Of the pyæmic, typhoid, and malarial types, the first two are met with far more commonly than the last; instances are very infrequent in which malarial attacks are simulated. The cases, on the other hand, are many that present themselves with irregular fever, inconstant in degree, and variable in daily range. Such "atypical" cases indeed constitute a remarkable feature of malignant endocarditis, and it is futile to attempt to classify every case of the disease according to hard and fast types, the resemblance to which is, at best, seldom more than superficial, and, as the disease is seen in practice, often altogether wanting.

Among other symptoms of a general nature is *headache*. This is sometimes of great severity, and, accompanied as it may be by *vomiting*, has on several occasions given reason for suspicion of meningeal trouble. Meningitis may indeed be present, but the diagnosis must be made with caution, since there have been cases in which during life meningeal symptoms were present, and even pronounced, and on subsequent examination the meninges were found healthy.

Various forms of *rash* have been observed in association with malignant endocarditis. The eruption may consist of a simple erythematous mottling, or it may be purpuric. It is not, however, a common feature of the disease, and must be distinguished from embolism of the skin, of even rarer occurrence, in which an area of redness is to be seen around a central dark hæmorrhagic point, the latter indicating the position of the blocked vessel; a small abscess may subsequently form and discharge itself, leaving a superficial ulcer.

Before leaving this group of symptoms, attention must be drawn to the importance of discriminating, if possible, between the pyæmic manifestations of malignant endocarditis and symptoms that owe their origin to the initial disease, to which the endocarditis itself arises as a secondary condition. The distinction is not always easy. In the latter event the disease is to be regarded as one of general pyæmia, with the various symptoms of pyæmia, and the various risks attendant on pyæmia,—among them the risk of secondary or metastatic infective processes, of which one, infective inflammation of the endocardium, has been realised. Thus, in the course of a general pyæmia, signs of cardiac disease may make their appearance, and at the autopsy recent vegetations may be observed

on the valves of the heart; such a case is one of pyæmia complicated by malignant endocarditis, *not* one of malignant endocarditis with secondary pyæmic symptoms.

In the former case the septic trouble that was the original cause of the endocarditis may have subsided, or may never have been prominent, and the cardiac condition may have become the most important feature of the case, and in itself the source of secondary mischief. A state of pyæmia may arise out of the infective endocardial lesions, and such a case is properly regarded as one of malignant endocarditis with secondary manifestations of pyæmia.

2. The *cardiac symptoms* are for the most part the mechanical outcome of the diseased state of the valves, and are similar to those produced by valvular defect of rheumatic or other origin, depending not so much on the essential nature of the endocarditis as on the orifice affected, on the degree of valvular mischief, and on the amount of compensation. On the symptoms and signs of the varieties of valvular disease it is not proposed to dwell. One or two considerations, however, deserve notice.

It has been said that the symptoms directly referable to the cardiac lesions often occupy an inconspicuous place in the clinical picture of malignant endocarditis. This statement holds good for a large number of cases in which the mechanical defect in the working of the valves is slight, and therefore not such as to give rise to prominent signs or well-marked symptoms. In these cases, notwithstanding the malignant nature of the endocarditis, direct evidence of valvular disease, such as murmurs and cardiac symptoms proper, is to a great extent obscured by the gravity of the constitutional disturbance, and the prominence of pyæmic symptoms. Signs of cardiac disease may even be completely wanting; and instances of malignant endocarditis, proved to be such by autopsy, have been recorded in which careful and repeated examination has failed to disclose any fault in the working of the heart.

The disease may, however, present itself under another and a very different aspect. Among its manifold guises is one that has been described as the *cardiac type*, in which the group of pyæmic symptoms is comparatively unobtrusive, and the patient has the aspect of one who is suffering, in

the main, from heart disease. The onset in such cases is usually insidious, and the course of the disease is prolonged. There is danger of overlooking the infective nature of the endocarditis, and of regarding the case as one of simple valvular defect. The distinction is sometimes difficult, and a diagnosis can only be arrived at after an extended period of observation. Help may be afforded by clinical evidence of the affection of a part of the endocardium not commonly involved in simple endocarditis. Thus there may be murmurs indicative of disease of the right side of the heart, at the pulmonary or tricuspid orifice. The positive value of such evidence is great, but it must be remembered that lesions on the right side of the heart are found *post mortem* in only about one in six of the cases investigated, and of these it must often happen that during life the physical signs were not sufficiently definite to justify the diagnosis of right-sided endocarditis.

Some stress has been laid on the appearance of fresh murmurs, while the patient is under observation, as affording evidence of a malignant endocarditis. Diastolic and systolic murmurs may be heard at a spot where previously a systolic murmur only was audible; or a murmur may arise in connection with an orifice of which there was previously no evidence of disease. It is easy to strain the significance of such an extension in the physical signs. In the first place, it cannot be inferred with certainty from the appearance of a fresh murmur that there has necessarily been an advance of structural disease. Without any change in pre-existing structural lesions, the development of a murmur may be a sign either of recuperation or of failure in the heart's action. On the one hand, a heart which by reason of feeble action is unable to produce a murmur at a diseased orifice may under appropriate treatment regain its strength, and with renewed force of contraction a murmur may be generated. On the other hand, the cavities of a failing heart may become dilated to such a degree that regurgitation of blood, and in consequence a murmur, arises at an orifice, the valves of which are structurally intact.

These two fallacies, if kept in mind, do not, as a rule, lead to difficulty. There is, however, a further consideration of greater importance. In the event of the appearance of fresh murmurs, not due to either of the conditions just mentioned,

it may be assumed with some confidence either that valves previously healthy have become diseased, or that disease has advanced in a valve originally affected. Now, from the fact that there is advancing disease, it is not justifiable to argue that an endocarditis of infective nature is present. A progressive endocarditis is not necessarily a malignant endocarditis. Simple chronic endocarditis may, and commonly does progress, and with its progress fresh signs are added to those that were previously observed. The process is, however, gradual, and as a rule interrupted by periods during which there is no evidence that the disease is progressing. Abrupt and repeated change in physical signs is significant of rapid and continuous destruction of tissue, and such sudden change from day to day is a feature of some cases of malignant endocarditis, but these are not the cases that present difficulties in diagnosis.

It is evident that the significance of a fresh murmur may easily be exaggerated, and that in itself it is anything but an infallible guide to the nature of the disease. Its value is, however, greatly enhanced if it be associated with other symptoms, such as continued fever or infective embolism.

3. The third group of symptoms comprises those of *embolic* origin, due to the detachment of fragments of vegetation from the diseased valves of the heart, and their conveyance by the bloodstream to distant organs, where they become lodged as emboli. Embolism may occur in complication of simple endocarditis, but it is far more frequent in the infective variety, and in this condition is often of the utmost value as an aid to diagnosis. The embolic fragments, in common with the vegetations from which they are derived, contain as a rule micro-organisms; and hence, being infective, they are apt to lead to infective processes in the tissues to which they find their way, processes that not uncommonly terminate in abscess.

Of various organs, emboli are more often arrested in the spleen and kidneys than elsewhere. They may, however, as has been stated, block the vessels of the brain, skin, intestine, retina, and other parts; or a branch of the pulmonary artery may be occluded by an embolus detached from the valves of the right side of the heart.

Embolism does not inevitably give rise to symptoms. The event is usually marked by a rigor, and followed by a rise of temperature. The occurrence of local symptoms is mainly determined by three factors, (*a*) the freedom of the collateral circulation, (*b*) the size of the vessel that is blocked, and (*c*) subsequent changes in the affected area. Some indication is necessarily afforded when the vessel is of such a size that its occlusion interferes with the proper function of the organ to which it should carry blood. For instance, an attack of pain in the side followed abruptly by breathlessness with rapid respiration may be the outcome of embolism of a branch of the pulmonary artery. Or the sudden onset of hemiplegia, complete or partial, may proceed from the occlusion of the middle cerebral artery or one of its branches. Other symptoms have reference to inflammatory changes that may follow embolism. Thus attention may be directed to the spleen by the complaint of pain in the left hypochondrium, and on palpation it may be discovered that this organ is uniformly enlarged and tender. The enlargement is the outcome of congestion; the tenderness is evidence of a localised peritonitis in the neighbourhood of the infarction, and probably due to a direct extension of the infective process.

In other instances the peritonitis is generalised, perhaps not so often from a gradual advance of infection, as due to the formation and rupture of an abscess, followed by the dissemination of infective material throughout the peritoneal cavity. Pleurisy, localised or general, and in the latter event likely to terminate in empyema, may similarly follow pulmonary infarction. Embolism of the kidney is marked by the appearance of blood and albumen in the urine, and may be the starting-point of an infective nephritis, as shown by the presence of pus, as well as of renal cells and tube-casts, in the urine. Embolism of the retinal artery may lead to an infective inflammation that may ultimately involve the whole eyeball in destruction.

Other forms of inflammation may arise, doubtless of similar origin, and proceeding from the cardiac condition, though not obviously due to the embolism of gross particles of vegetation. Among these are (*a*) inflammation of the serous membranes, pleurisy, pericarditis, and peritonitis; (*b*)

inflammation of the joints; and (*c*) meningitis. It has already been described how peritonitis or pleurisy may originate by extension of inflammation from a neighbouring septic infarct. The peritoneum, pleura, or pericardium may, however, become inflamed without obvious infarction of underlying tissues, though no doubt by the conveyance in the blood-stream, somehow or other, of infective micro-organisms.

Septic arthritis is not a common complication of malignant endocarditis, which in this respect is in contrast to the ordinary forms of pyæmia. Suppurative meningitis may supervene in the course of the disease, and is an indication that the fatal event will not be long delayed. There is, however, always difficulty in making sure of the onset of meningitis, since meningeal symptoms may be present, and indeed marked, and yet the meninges may be found to be free from disease.

From the foregoing account it is evident that malignant endocarditis is a disease that comes before the clinician in many different forms, and that one of its leading characters is a want of uniformity in clinical detail. In onset it is in noted cases abrupt, in other cases insidious. In course it is sometimes rapid and tumultuous, with acute symptoms, and almost bewildering by reason of abrupt and repeated change; at other times it is slowly progressive, with periods of comparative quiescence. Of the various clinical forms some may bear a likeness to other states of disease, and this has led to the differentiation of certain "types" of malignant endocarditis. Typhoid, pyæmic, malarial, meningeal, and cardiac types are among those that have been described; but it is urged that such a distinction is in many cases an artificial one; that the resemblance is only in rare instances so close as to involve great risk of error; and that it is characteristic of malignant endocarditis that a large number of cases present irregular and atypical features, bearing no close likeness to any definite type of disease.

The vagueness of the symptoms is often a cause of great uncertainty in diagnosis. The possibility of a continued fever without obvious cause being due to malignant endocarditis has always to be considered. Other conditions suggested by the symptoms may be excluded one by one; but a diagnosis that is based mainly on exclusion is

necessarily unreliable, and clear indications of a positive value must be present before the diagnosis of malignant endocarditis can be made. Such evidence is to be sought for in the state of the heart and the occurrence of embolism.

The diagnosis consists of two steps. In the first place, it must be clear that there is endocarditis; and secondly, there must be evidence that the endocarditis is infective. The significance of sudden and repeated changes in the various signs of valvular disease has been discussed, and it has been pointed out that such change is suggestive, though not in itself sufficient proof of a malignant endocarditis. It has been indicated that evidence of much greater value is to be found in the repeated occurrence of embolism, particularly if followed by septic infarction; in the latter event there can be little doubt as to the nature of the disease. The association of valvular disease recurring embolism, and fever for which no other adequate cause can be found, is sufficient for the diagnosis of malignant endocarditis. There are, however, many cases in which, although malignant endocarditis be suspected, the clinical evidence is not sufficiently definite to justify a diagnosis; clear indications may be wanting even up to the time of death, though afterwards there are found the lesions of an infective endocarditis, well marked and even advanced.

Treatment.—Of treatment there is unfortunately but little to be said. No means have yet been discovered that are efficient in staying the course of the disease. Cases are recorded in which the diagnosis of malignant endocarditis has been made, and recovery has ultimately ensued; but in these the very fact of recovery affords ground for reasonable doubt as to the accuracy of the diagnosis, and at the same time renders confirmation impossible.

It is possible that malignant endocarditis may eventually find a place among the affections that are amenable to serum methods of treatment. But in this connection it must be borne in mind that malignant endocarditis is not a specific disease. As there is strong reason to believe that the same form of micro-organism is not in every case the cause of the disease, so it is not to be expected that a specific mode of treatment, applicable to all cases, will be discovered.

Anti-streptococcic serum has been employed, but so far without encouraging results, and the

treatment resolves itself into the maintenance of general nutrition by every possible means. Nourishing and easily digested food is essential, and quinine may be given with benefit, in combination with iron. Stimulants are indicated at an early stage. But, when the diagnosis is established, no more can be expected of treatment than to postpone the termination of a disease that must ultimately prove fatal.

A PLEA FOR A MORE PRECISE CLASSIFICATION OF DISEASE.*

BY

W. J. TYSON, M.D., M.R.C.P.Lond.,
Senior Medical Officer, Victoria Hospital, Folkestone.

THE time seems to me to be approaching, if not already at hand, when a more definite and diagnostic classification of diseases is required. The causes of disease are becoming better known every day, and many that in the past have done duty, either under the head of "predisposing" or "exciting," must fall away and give place to others, more precise and causative. Until a few years ago, a cold or a chill was thought to be the cause of nearly all inflammatory states in every part or organ of the body; it acted as a satisfying and comfortable explanation of all troubles, alike to patients, their friends, and the doctor; it covered up an immense amount of ignorance and spared need for further clinical research. But in these last days of pathological, clinical, and laboratory examination, cold as an origin of disease must take a declining position; that it plays some part in disease, although at present it seems to be a mysterious one, we can hardly doubt, but as time goes on the importance of the above cause will become less and less as knowledge advances.

Another cause which in this part has played great havoc among mankind is that which went by the names pyæmia, hospital gangrene, &c., which has gradually become less and less virulent, until it has almost disappeared as it was once known. Still, what for want of a better name at present may

* Read before the members of the West London Medico-Chirurgical Society.

be called a septic origin of disease, will, I think, for some time to come occupy a prominent place. It has practically disappeared as a result of operative surgery, or in other words, the introduction of a septic origin of disease from without can be prevented in the surgical work; but in a large number of cases pus or matter becomes pent up in inaccessible or undiscoverable positions, so that septicity will long remain a cause, and must always be thought of when searching for some unexplained high temperature.

No doubt as our laboratory work extends there will be a defining division in what is commonly called septic disease—fresh bacteria discovered; still for present purposes the three or four well-known bacteria sufficiently indicate, when present, what is meant by a septic origin of disease.

Another common cause of disease, and one of the most important, is the presence of the bacillus tuberculosis with its attendant inflammatory changes. Tuberculosis clinically in its ordinary phases is well known, and as a rule there is not much difficulty in arriving at a diagnosis.

It is within my recollection that many of the cases of hip disease in children were thought to have their origin in blows and falls, which with our present knowledge would without hesitation now be classed as tuberculous.

To come down to more details. Words are constantly being used for diseases which are meaningless, and carry with them no true diagnosis, but only a rough pathological condition, and not indicating any particular line of treatment.

Let us take the word pneumonia; under this head is generally included what is termed acute croupous pneumonia, catarrhal pneumonia, and, thirdly, interstitial pneumonia. Formerly pneumonia practically meant only one form of the disease, viz. the sthenic form, with all the well-known classical symptoms and signs, and for which bloodletting was commonly done. I believe that this was the only form of pneumonia known to our forefathers, and when the word pneumonia was used it carried with it all those symptoms and signs that we generally associate with the disease now known as acute croupous pneumonia occurring for the most part in healthy young adults. If there is one disease which seems to be caused by a chill more than another this is certainly one, yet it must be mentioned that many

cases occur when no such exposure has taken place.

I would then advocate that if the word pneumonia is to be retained, standing alone without an accompanying adjective, that the acute croupous form should be always understood when the above word is used. But to use the word indiscriminately for all sorts and conditions of inflammatory states of the lung is a misnomer, and leads to errors in diagnosis and treatment.

The pneumonia occurring in influenza, known to all of you, is clinically quite distinct; it has its own history, its own peculiar features, runs a certain course, and is practically only a symptom of the general disease. It is, in fact, only one of many results that may be met with in influenza.

Take again that form of pneumonia met with in alcoholic persons, the leading symptoms of which are moderate temperature, excessive expectoration, spreading of the inflammatory condition, great depression of the nervous system, low circulatory condition, &c.; or again, that infective form of pneumonia which comes on—perhaps days or even weeks—after erysipelas, characterised by small patches affected, wandering in character, quickly appearing and afterwards disappearing, resistful to treatment, fatal in prognosis; or again, that form which occurs late in typhoid or typhus; and my list might be extended, but I have said sufficient to indicate that each of the above forms are unlike, have a different origin, run a different course, and requires a separate or a distinct line of treatment.

Peritonitis.—It appears to be very questionable whether this word should ever be used without some explanatory adjective. Has any one here ever seen a case of so-called simple peritonitis? All my life I have been looking out for such a case, and so far have not met with one. The causes of peritonitis are comparatively few in number, and I hardly think that "cold" can be reckoned among them.

In children and young adults, apart from typhoid fever and tuberculosis, it is almost invariably due to disease of the appendix. I have met with it from injury and also from strangulation of the gut in connection with Meckel's diverticulum and intussusception, but from chill and blood-poisoning never. Coming on to middle and later life, one must add to the above a few more causes, such as

propagation of inflammation from other organs, viz. ulceration or perforation of the stomach or bowel, strangulation of the bowel, metritis, &c., new growths in the peritoneum, and almost lastly, peritonitis from injury to the peritoneum, produced by accidents or from operative procedures.

We have all seen exceptional causes of peritonitis; there is that somewhat rare condition, happening in late life, of falling of the splenic flexure of the colon upon itself, at first setting up a flatulent condition of the bowels, constipation (the constipation being a common cause of application for medical advice), the attacks, at first mild, gradually increase in frequency and in length, until ultimately a peritonitis may close the scene: such a case I made a post-mortem upon a few months ago. These cases no doubt would be benefited by the treatment that has been recommended by Mr. Hutchinson, by elevation and massage.

Again, there are a few cases recorded which may be called provisionally septic peritonitis, from absorption of material outside the peritoneal cavity, such as from the urethra, &c. I have met with infective arthritis in small and large joints, iritis, pleurisy, pericarditis produced by a gonorrhœal discharge, the peritonitis associated with gonorrhœa in women, is well known, but I have not seen the association of gonorrhœa with peritonitis in men.

As I have doubted the existence of simple peritonitis, so I do that of simple meningitis; I am bold to confess that the only three causes of meningitis in children known to me clinically are tuberculosis, extension from ear disease, and blows to cranium. One would think that the disease was produced by many causes, considering how frequently the word is in use. The long list of distinguishing symptoms and signs between simple and tuberculous meningitis exist, I think, to a great extent, in the author's own brain. Coming on to middle life, other causes of meningitis have to be remembered, such as syphilis.

I have now been through the common clinical causes of pneumonia, peritonitis, and meningitis; my object has been in these few short notes to emphasise the importance of always, or nearly always so, when using the above words, of placing a diagnostic adjective in front of them. I am quite aware that under the above words of pneumonia, meningitis, orchitis, cystitis, &c., the causes or

origin of the disease is given in our ordinary text-books, but there is want of proportion about them, and the common and rare causes are mixed or jumbled up together, and, like a tangled net, the threads (or causes as it may be), require much trouble to unwind them.

Those of us who have been in practice for a few years, or hold hospital appointments, find out sooner or later what value to put on the causes of disease that are commonly given in books or elsewhere; but for those starting in practice, or those who are in practice in out-of-the-way places, and have not such advantages as some of us possess, would fail to a considerable extent to put that due value on causes which belongs to them. There is another reason why I believe rare causes of many diseases have a far too great importance attached to them; our medical societies, especially perhaps those in London and the great provincial towns, or perhaps it would be correct to say that the men who write for them, wish to say something that is not commonplace, and so all rare cases are hunted up and promptly recorded; these are published in the medical journals, and thus obtain a prominence and an amount of weight out of proportion to their clinical value; whereas our common causes of diseases are more or less neglected, and so there is a tendency to forget the well-known and useful aphorism, "Common things most commonly happen," or we rarely meet with rare diseases.

Again, when using the words above singly, there is a tendency to rest satisfied. Peritonitis, meningitis, cystitis, I maintain is not a diagnosis, but only a symptom, although, of course, a most important and possibly vital one; yet these symptoms are placed in all text-books at heads of chapters, and are practically treated as entire diseases.

This is not a fancy point I am calling attention to, for after all treatment, which to the patient is everything, is affected most materially according to which adjective we employ. A tubercular meningitis had better be left alone; a septic meningitis requires probably active surgical treatment; appendicular peritonitis, if local in position, is cured generally by local surgical treatment, &c. &c.

The ordinary sthenic pneumonia is not the same disease clinically as septic pneumonia, influenzal, or alcoholic pneumonia, and the treatment in each, I take, would be somewhat different.

My paper advocates rather a clinical classification of disease than a pathological, and this is not to be wondered at, seeing that my work is almost entirely confined to the bed-side rather than the laboratory, whereas those engaged in physiological and laboratory studies would make bacteriology the prominent factor in classification. The assistance of both the clinician and bacteriologist is required, and they must work in harmony with each other. Still, just at present I think the clinician has fallen too much in the background, and requires a little push forward.

NOTES.

Tetany in Childhood.—Hauser ('Berlin. klin. Wochenschr.,' 1896, xxx, 782) said that although the study of tetany in the adult stood on a firm basis, this could not be said of our knowledge of tetany in children. Even Strumpell thinks that the peculiar convulsive conditions which occur in little children should not be identified with tetany. The general belief is that the diagnosis of tetany in children must be based upon the same points as in adults. Escherich, in 1890, brought the disease in direct relation with the familiar and frequently observed spasm of the glottis. Loos, Escherich's pupil, even went further, and formulated the following propositions :

(1) The tetanic contractures of the extremities are rare in the tetany of children, and are not essential symptoms.

(2) Spasm of the glottis, however, is almost a pathognomonic symptom. There is no proof that a laryngo-spasm exists without other symptoms of tetany.

(3) Tetany stands in no causal relation whatever to rickets.

The author, from a study of 280 children, comes to the following conclusion :

(1) There is a *true tetany* of childhood which follows the exact symptomatology of the tetany of adults, and presents, as the most marked symptom, the characteristic contractures of the extremities ; in addition, there is always found one or the other, and frequently all the symptoms of the so-called *trias*. Aside from the tetanic spasm, the laryngeal and eclamptic spasms play an important, even a

prognostic rôle. The ætiology of infantile tetany is not yet clear. It is a fact of experience that tetany rarely attacks perfectly healthy children. Rickets seems to be an important predisposing cause. The direct exciting cause seems often to be gastro-enteric disturbances. This is shown by the frequent concurrence of tetany with acute dyspepsia. The tetany of childhood is a dangerous malady, frequently causing death. Its therapy can, in the present state of our knowledge, be a causal one only in those cases where more or less disturbance of digestion is present. These cases should be treated by a rapid emptying of the digestive tract by irrigation of the stomach and bowels, the administration of purgatives for the removal of the toxins, and to avoid their new formation the restriction of the diet to amylaceous food. In all other cases the treatment can only be a dietetic one. Symptomatically it resolves itself into the administration of narcotics (bromides, chloral hydrate, &c.) for the spasms.

(2) Besides this form of tetany characterised by typical contractures of the extremities, there is a so-called *latent form*, occurring in infancy. Its diagnosis is based on an increased galvanic irritability of the nerves, which does not occur in this manner in any other disease ; the same may be said of Trousseau's phenomenon, if present. A well-developed Chvostek's sign may support the diagnosis, but is not convincing in the absence of one of the other symptoms.

(3) These cases of latent tetany frequently present as a prominent symptom severe spasm of the glottis. All cases of spasm of the glottis, particularly those of a severe type, should stimulate us to look for tetanic symptoms, especially for Erb's and Trousseau's signs.

(4) The greater number of all cases of spasm of the glottis have no connection ætiologically with tetany ; moreover, some relation must exist between spasm of the glottis and rickets, which probably indicate more than a mere coincidence.

Pediatrics, April 15th, 1897.

Veratrum Viride in Eclampsia.—Dr. W. H. Thayer claims that in puerperal convulsions he has found a condition of the nervous system that begets a peculiar tolerance of certain drugs, especially of veratrum viride, so that the officinal dose has no effect. But large doses quiet the

nervous erethism, producing a decided effect in a short time—sometimes in fifteen minutes, but almost certainly within an hour—and keeping the nervous system under control for several hours. The administration is followed by cooling of the surface, great lowering of the pulse in rate but not in strength, and along with this complete arrest of the convulsions. The state of the pulse is the guide in treatment. From a high rate, which rules in the disease, it is reduced to the normal standard or below it; and while it is kept below 60 there need be no fear of a recurrence of the paroxysms. When this effect has been once produced, it will continue several hours, and a single dose will do it; if not apparent within an hour or less, the medicine must be repeated in smaller doses, and it can be safely repeated at intervals until the pulse begins to fall. With the pulse for a guide, no untoward symptoms need occur from its use; the pulse may be brought to 50 without any general depression; if carried so far as to produce vomiting we may find great prostration produced by the nausea, which is overcome within thirty or forty minutes by opium or any diffusible stimulant, perhaps in less time by a solution of morphia hypodermically. The employment of veratrum viride in large doses in puerperal convulsions was first reported to the King's County Medical Society by Dr. Herbert Fearn, of Brooklyn, in 1869, substantiated by cases in which he had used drachm doses of the tincture, sometimes required to be many times repeated, with favorable results. Since 1869, when Dr. Fearn's paper was read, the treatment of puerperal convulsions with veratrum viride has been slowly gaining favour, of which we have evidence in many communications to various societies and medical journals. Bartholow, in his 'Materia Medica and Therapeutics' (1888), says: "Dr. Sullivan, of San Francisco, informs me that veratrum viride (half a drachm of the fluid extract every fifteen minutes, until nausea or vomiting ensues) is invaluable in puerperal convulsions. Barker, in his 'Puerperal Diseases,' has already called attention to its utility, and Boyd confirms the previous observations. Increasing experience adds to the testimony regarding its exceptional value in relief of this formidable malady." "The experience of one general practitioner in puerperal convulsions is not very great; but in the convulsions of children I have regularly employed

veratrum viride for twenty years past with excellent effect, and as a prophylactic in ephemeral fever in children who are subject to convulsions. I believe the use of veratrum viride in puerperal convulsions originated in Brooklyn, where its value is now generally recognised. It has gained ground elsewhere, as rapidly as could be expected of the management of an affection so infrequent, and promises in time to supersede all other methods of treatment."—*Boston Medical and Surgical Journal*, April 1st.

Sulphonal in the Treatment of Night Sweats.—Combemale and Descheemaker are stated by 'Revue de Thérapeutique Médico-Chirurgicale,' of December 1st, 1896, to have obtained excellent results from the use of sulphonal in the night sweats of phthisis. From fifteen to thirty grains of the drug are given each night. The cough is also diminished. In the very advanced stages of tuberculosis, however, the influence of this drug over the sweat is not marked.

Therap. Gaz.

The Wearing of Veils and its Effect upon the Eyesight.—(1) Every description of veil affects more or less the ability to see distinctly. (2) The most objectionable kind is the dotted veil. (3) In undotted and non-figured veils, vision is interfered with in direct proportion to the number of meshes per square inch. (4) The texture plays an important part in eye-strain produced by the veil. When the sides of the mesh are single, compact threads, the eye is embarrassed less in its effort to distinguish objects than when double threads are employed. (5) The least objectionable veil is that without dots, sprays, or other figures, but with large regular meshes made with single compact threads.

Charlotte Med. Journ., April, 1897.

Red Gum (B. W. & Co.).—This well-known drug, long known as a mild styptic and antiseptic and as an excellent local and general astringent, has been prepared in "tabloid" form for some time. Recently improvements have been effected in the formula. When a Red Gum "Tabloid" is allowed to dissolve slowly in the mouth, the continuous action of the drug on the mucous membrane of the throat is fully secured.

THE CLINICAL JOURNAL.

WEDNESDAY, MAY 19, 1897.

DEMONSTRATION OF CASES AT WEST LONDON HOSPITAL,

March 17th, 1897.

By DR. WHITFIELD.

GENTLEMEN,—I wish to show you first a very simple but extreme case of pleurisy, in a man *æt.* 40, whose occupation is that of a billiard marker. The history is that two months ago, after coming out of a hot billiard room, he caught cold, and soon afterwards noticed a sharp pain under the heart; he, however, continued at work, but six weeks after the first attack he noticed that his pain suddenly became very much worse, and was aggravated by breathing and coughing. He went to bed, but did not consult a doctor. A week ago he felt slightly better and returned to work, but the pain returned and he became very short of breath, and continually felt giddy and faint. He was brought into the hospital on the 9th, when the whole left side of his chest was found to be completely dull. No breath sounds were found to be present, and no voice sounds, but there was whispering pectoriloquy to be heard at one point. When he was laid flat upon his back the cardiac dullness was on the right side of the sternum, and the impulse was felt in the fourth right interspace, internal to the nipple line. A few friction sounds could be heard at the right base. There was an enormous effusion in the whole of one side of the chest, and the heart was thrown over to the right side. Where there are these enormous pleurisies the pericardium is thrust over; but in most of the autopsies on pleuritic cases which I have made, it has not been rotated as is generally stated. This patient has had 34 ounces drawn off, but the effusion has not by any means disappeared, and the chest is practically dull right up to the clavicle and to the middle line. I have examined his heart sounds to-day, and find that the heart is on the way to return to its normal position. I can detect

no apex beat, and that is a usual event with a heart which has been driven over to the other side, for at one period of its return it gets behind the sternum. The left side of the chest is less moveable than the right side. No breath sounds are heard below the level of the second interspace, and the voice is only heard, I think, through the ear and not through the stethoscope. The whispering voice is still heard, but not so loud as it was. There is a suggestion of bronchophony; I take it that that is due to solidity of the lung—a patch of consolidation behind the fluid.

Acute pleurisy is divided, as a rule, into acute dry pleurisy and acute pleurisy with effusion. I think acute dry pleurisy is practically subacute pleurisy; as a rule, the temperature in this form does not rise so high as in the effusive variety. Plastic lymph comes out, but is followed by no fluid, and the case runs a more chronic course. Probably the lung in every case becomes adherent where the lymph has exuded, and unless a man has frequent attacks, that is the end of his trouble. If the patient is unfortunate enough to have repeated attacks, he probably gets a chronic adherent lung. In post-mortems of people over fifty it is quite exceptional to find a lung where there is no adhesion anywhere on its surface, and in many of these cases there is nothing to show that the patient has had to lie in bed for pleurisy.

In acute pleurisy with effusion we have points of very great interest. We may divide them into cases of acute pleurisy coming on idiopathically from chill, and cases coming on in the course of acute fevers, chiefly rheumatic fever. Some years ago, some professors in France and Germany made some suggestions on the subject, from which it appears that in the vast majority of cases of so-called simple "caught cold" pleurisies the affection is tubercular, although in most cases it would be difficult to find the tubercle bacillus. Still this latter fact does not stand for much, because in many of the undoubted tubercular manifestations you may search very diligently without finding the particular microbe. But the after history of these cases showed that if the original disease was not

tubercular, tubercle became an element in the later history. Curiously enough, this tubercular element was not confined to the "catch cold" pleurisy, but was found in several pleuritic patients who died, and in whom the pleurisy came on late in the course of chronic Bright's disease, heart disease, and sometimes occurring with cancer, not necessarily cancer of the pleuræ, but cachectic cases, which wound up with an attack of pleurisy. These facts should make us careful when giving a prognosis in cases of acute pleurisy without any known cause but chill. Though a patient suffering from acute pleurisy has, as a rule, a far better chance of recovery than a subject of acute croupous pneumonia, you will find that the pneumonia patient has not had the faintest trouble afterwards, but that the pleurisy patient has.

The next case is very interesting to contrast with the one we have just looked at, because it is that of a girl who had pleurisy at 16 years of age. She is now 21 years of age, so that the case is exactly the converse of the last. The right side of the chest is obviously larger than the left. Loss of movement occurs in both cases, in one case the lung is partly crushed, and in the present one the loss of movement is due to strapping down. There is no fluid in the chest of this girl at present, and from the history she had a common "caught cold" pleurisy, and I think I can give you evidence to show that she is now becoming tubercular, no doubt owing to the fluid having been allowed to stay too long in the chest when she had her attack, and thus crippling the lung. She also has a violent attack of herpes zoster on the right side, which I have not been able to find any cause for. There is some sign of affection in the other lung, and there may be some pleurisy at the back of it. We now know that herpes zoster is very often the result of compression of one of the intercostal nerves, or hæmorrhage into or pressure on the ganglion, and it is possible that in cases of pleuritic trouble these nerves or the ganglion may be interfered with, as they lie so close. Some of the places are scarring. You will find that her apex beat is very distinctly felt rather behind the anterior axillary line. She states she was in bed three months when she had pleurisy, and that the chest was tapped. You will find that there is very little movement, and that on percussion at the back

there is complete dulness practically right up the back. There are a few friction sounds in the position of the normal apex beat. There is definite bronchophony, so that one might take it for a very large cavity. There are also a few signs of softening and some fine crepitations. The expectoration is not purulent.

I think these two are very analogous cases. They are both left-sided pleurisy. The man has probably had his to a greater or less extent for two months. I should be inclined to think that the effusion was not great until a week before he came in, because he gives a very distinct history by saying that besides the pain he felt very giddy and faint. I cannot understand a man with so much effusion going about his work as this man appeared to do, although it is true a billiard marker's work is not very laborious. In the man we have the chest full of fluid on one side, the pleuræ roughened, the heart pushed over to the right side, and the lung almost completely collapsed; there is no high-pitched note at the apex, showing the compensatory emphysema which is common in pleurisy, I think for the simple reason that the effusion is so high that it covers the apex as well as the rest of the lung, leaving no room for emphysema. When the fluid is absorbed or drawn off, one of two things happens—either the lung expands to meet the chest, or the chest is to a certain extent drawn in. There is no doubt that the early drawing off of a pleuritic effusion, and seeing that a large effusion that is crushing the lung does not remain too long, is one of the chief points in the treatment of a large pleurisy; because if the lung is compressed for long, as it is in the man I am referring to, it tends to lose its elasticity, it gets enormously congested, the circulation through it is impeded, all the capillaries and small arteries are distorted, and the lung becomes atrophied. After that, if one draw off the fluid the lung cannot be got to expand properly, and if a great deal of force be used a part of the lung might be ruptured. When aspirating with very little pressure, the fluid may cease to flow through the needle before the liquid has all come away, for the simple reason that the negative pressure in the chest equals that on the outside. Then the lung touches the chest wall in some parts only, not in others, and in those parts fibrous adhesions

form; this may give rise to sounds similar to those noticed in the girl who was before you. That is to say, the pleura, instead of being a fine serous membrane, becomes thickened, and from it strands of connective tissue dive into the lung, analogous to what one sees in granular kidney, in which strands go into the pyramids and distort the kidney. These strands then begin to pull on the lung, and the lung gets stretched. Now it is obvious that one of two things must happen; either the chest must fall to meet the lung, or the lung must expand to meet the chest wall. I have no doubt that in most cases both processes occur. I believe the lung partially expands, but it never again becomes true lung tissue, being enormously fibrosed. Apart from that, the bronchi, which are of course filled with air, practically never get collapsed. The air can enter the bronchi freely at normal atmospheric pressure, while outside they are subjected to the traction of the fibrous bands, and the negative pressure from the elastic chest wall. I feel sure, if you were to see the lung of the girl in section, you would find a large number of small bronchiectases or small bronchial dilatations, and that would account for the enormous vocal resonance and pectoriloquy. The cavernous breathing in this case is not produced by large cavities, but by many small ones.

There is no phthisical history in the family of the girl, and she is not very phthisical-looking; she does not sweat at night, and she is well nourished. She says she is not wasting. But those who listen to her lung would notice that she has catarrh, at all events in the alveoli at the right apex. She has some fine crepitations, and they have been extending somewhat slowly while she has been under observation. I feel no doubt that this case is going to turn into a tubercular one. In the future we must look upon this, therefore, not simply as a case of interstitial pneumonia following pleurisy, but one developing into fibroid phthisis. That may be the future history of the man in the ward. He will be very carefully looked after here, and we may succeed in getting his lung to expand, but I rather doubt it.

As regards the treatment of the first case, acute pleurisy, there are very few points, but they are very important, and are very much discussed. As regards the pain of pleurisy and the harassing dry cough, I think the best drug to give is opium.

There is no fear of locking up large quantities of secretion, the cough is a very nervous one, the pain is very acute, the drug quiets the heart, and the patient is in the best condition for getting over his attack. The effusion is the next point to consider. It is obvious that until the effusion is there, all one has to do is to keep the patient in bed and comfortable by means of opium. Another beneficial measure before the effusion comes out is to strap that side of the chest just the same as one would strap a broken rib. Very often that proceeding causes almost complete relief of the pain. When the effusion comes I think the strapping should be removed. With the appearance of the fluid the pain stops, or nearly so, because the fluid comes between the surfaces which were rubbing together. It would therefore seem a pity to draw off the fluid and bring the surfaces together again. But here we must be guided by the amount of the effusion. If the amount is only small, one would not think of drawing it off. Sudden death is not uncommon in pleurisy. It has been noted in many cases, more especially left-sided, which in my experience is more frequent than right-sided. I think one may very much lessen the number of those sudden deaths by not allowing the fluid to reach too large a volume.

We used to be taught two things in connection with this: first, that we must never draw off the fluid until the temperature is down, and secondly the fluid must never be drawn off in tubercular cases. But according to modern ideas both these injunctions are incorrect. We do draw off the fluid if it is embarrassing, whatever the circumstances and whatever the temperature. We also now know that most pleurisies are tubercular, so that the direction must be modified into "Don't draw off the fluid if there is obvious tuberculosis of the lung." If the latter be the case, of course the drawing off the fluid might result in a considerable spread of the disease. In aspirating, if the patient is nervous the pain can be got rid of by a cocaine injection beforehand. But in an ordinary male patient it is sufficient to hold a lump of ice for three or four minutes against the part through which the needle will be pushed. As to the amount of fluid to withdraw, it is important not to lay down a hard and fast rule. In the case in the ward we drew off thirty-four ounces with

apparently no effect except to increase his comfort and reduce the pressure. I have drawn off as much as eighty ounces in a case at one aspiration, but I did harm by taking too much, as the patient had a very violent fit of coughing in paroxysms lasting for one and a half hours, so that I had to keep him up with brandy. I think the great point is to judge by the pulse and the presence of cough. At the first cough I should recommend you to withdraw your aspirator from the chest, whether the cough be apparently accidental or due to the needle. The first cough is the herald of more coughs, and although I believe it to be usually a safe cough, if the patient's circulation is in a bad state it shakes him very badly and may terminate fatally. The man from whom I withdrew eighty ounces came into the hospital with a very irregular intermittent heart and advanced mitral disease. After he had been in the hospital about a fortnight he developed pleurisy very badly on the left side, which pushed his heart over to the right side. I determined to aspirate the chest until I brought the heart back to its normal position. But I have since learned that the heart takes time to come back. However, I nearly emptied the cavity of fluid. Within three hours, although the patient had been so distressed with his cough, he was as well as he had been before he had the pleurisy. Therefore I take it that the cough is more alarming than dangerous. With a small or even a moderate effusion one does not draw off the fluid in the early stages of the disease. But in any case if the effusion lasts a fortnight or three weeks I should draw it off. By leaving the fluid in, you are giving the fibroid changes a better chance of taking place. In most slight cases, however, it is unnecessary to aspirate. I should advise you to change the diet from that which the patient has been having during the acute stage. In the acute stage the patient should be given beef tea, milk, and milk pudding. Afterwards, however, as the condition is no longer inflammatory, I think he should be put on a modified Tufnell diet, controlling the amount of fluids. You will find it not very difficult in this case to get the amount down to a pint a day. If the patient complains of thirst, this will be relieved by giving small quantities of ice to suck. By thus reducing the amount of fluid in the blood, the blood will readily suck up the fluid in the pleuræ.

That is not only sound theoretically, but it is practically a very valuable method, and it is one I have always seen employed by Yeo in cases where he had any difficulty in getting the fluid absorbed, and that authority has been as successful in his treatment as anyone could wish. Next, it is desirable to increase the excretion of water, which is best done by means of salines, a morning dose of simple Epsom salts or compound jalap powder; half a drachm or fifteen grains of the latter in the morning will cause two or three loose evacuations, and get rid of a lot of water. Diuretics I am not in favour of, because they are apt to leave one in the lurch. Almost directly a patient gets any interference with the circulation, which of course a pleuritic effusion is, though not so great a one as cardiac disease, one seems able to administer diuretics to almost any extent without producing the desired effect. As it is immaterial in what way the water is taken away, I think it is preferably done by the bowel. Diaphoretics have been used, and I think that they are of some value. Pilocarpine has been used for the condition also, but I should be afraid to try it unless I felt it to be very necessary, because it is known to be an extreme cardiac depressant. In the case of the girl, it is far too late to do anything active. I do not believe from the signs that she is a subject of very malignant tubercle. Her other lung is undoubtedly emphysematous. The right lung extends over the middle line, which is common in these cases, compensatory emphysema having brought it across. I am convinced that the prognosis in post-pleuritic fibroid phthisis is not very bad. The best thing we could do for her would be to send her away to a tubercle-free atmosphere, such as the Swiss Alps, where her lung might undergo some slight expansion. I have in my mind the case of a sailor in the Navy who jumped overboard to save a fellow sailor in Plymouth. He saved the man, but got pleurisy himself. I saw him first in 1890, when he had very similar signs to the girl who has been before us. We procured him some Charity Organisation letters and got him over to the Cape. He had a bad attack of some fever, I forget what, and was sent home. A few years later I saw him again, and as far as I could remember he was in the same state as when he went four years previously. This seems to show that these cases exhibit a good deal of resist-

ance to the inroads of the bacillus. I have talked to the girl I showed you to-day, and pointed out the benefits of going away, but she is a servant girl and cannot manage it, and I therefore fear we shall be unable to improve the crippled lung.

I wish now to show you a case of phthisis with very interesting heart-sounds. It is an ordinary case of tuberculosis of the lung. With the first sound of the heart, at the base there is an undoubted abnormal exocardial sound. He has no other signs of pericarditis, but he has signs of pleurisy. I believe it to be a case of similar nature to one I saw at an autopsy, in which a case of apparently pericardial murmur during life turned out to be one of "peripericardial" murmur, that is in which the heart moved the pericardium against the lung, and there was pleurisy between that surface and the pericardium, where the murmur was produced. Of course one can modify it more or less by respiration, but it cannot be stopped. I do not think this is a sign of very great value, because most heart-sounds are modified by respiration.

Next I wish to show you a boy, a marked case of mitral disease, with clubbing of the fingers. It is not a congenital case, and there is no history of acute rheumatism, or of any disease exciting endocarditis, and this is quite common in young children. I generally find, by accident, that they have heart disease, and that was the case here.

This boy has a very interesting history in view of the relation of endocarditis to acute rheumatism, and in view of the relation of rheumatism to tonsillitis. He has cardiac disease, and has had repeated attacks of tonsillitis, and has pains about the joints, but he says he has never been laid up with pains in his joints at any time. The older physicians always drew great attention to the colour of the hair in rheumatism, saying that rheumatism was mostly associated with red hair. It is true that one notices a good many cases of rheumatism in which the patient has red hair, but there are many cases of rheumatism, and many children have red hair, so that there may not be much in that. The association is present here. He has the capricious appetite, pains in the legs, and wasting. At first I got very suspicious about these cases of wasting. I would like to ask you to always examine the heart whenever pains are complained of in the joints. They often come

with "growing pains," with a little cough, and you can find some fine crepitations which at first make one suspicious of tubercle. The murmur in this case is not much more than a week old.

ON SOME PAINFUL AFFECTIONS OF THE FEET.

By A. H. TUBBY, M.S.Lond., F.R.C.S.Eng.,

Assistant Surgeon to, and in charge of, the Orthopædic Department, Westminster Hospital; Surgeon to the National Orthopædic Hospital; Surgeon to Out-patients, Evelina Hospital for Sick Children.

PART IV.

Bunions.

THIS affection is also known by the synonyms of hallux valgus and hallux extrorsus. The term bunion is scarcely correct as applied to this affection, since the real cause of the trouble is the faulty direction of the great toe, and the bunion is merely the enlarged bursa which forms over the head of the first metatarsal bone and base of the first phalanx; but we will use the popular term bunion as indicating both the affection of the bone and that of the soft parts. Now, bunions are largely due to the use of improper boots, not necessarily of tight ones, but those which are pointed and often too short. In some cases it may be traced to osteoarthritis and gout. These diseases, however, merely give a faulty direction to the great toe, which is accentuated by narrow and short boots. The proper direction of the great toe has often been discussed, but there can be no doubt that in persons who go barefooted, the great toe and inner border of the foot form a right line. In some cases the great toe is even slightly adducted, and this is more marked among savage races, who use their feet as prehensile organs. Abduction of the toes is an artificial condition, and arises from wearing boots, and is exaggerated by the demands of fashion.

The feature of the deformity is the displacement of the great toe outwards, with projection of the base of the proximal phalanx and the head of the first metatarsal bones (Fig. 19). Both these portions of bone are often enlarged and covered with a false

bursa. The enlargement is especially noticeable in cases of osteo-arthritis, and I have on several occasions felt grating in the first metatarsophalangeal articulation. The anatomy of the affection is as follows:—The great toe is displaced outwards so as to leave a part of the head of the metatarsal bone uncovered, and a subluxation occurs with the result that the internal lateral ligament is stretched, and is in some cases thinned. The external lateral ligament is shorter and thicker than normal; those muscles the tendons of which are inserted into the inner side of the base of the first phalanx are stretched, those which are inserted into the outer side are contracted; on the dorsum, the tendon of the extensor proprius pollicis is displaced externally, and by its new position takes

The symptoms are sufficiently plain. In most people some displacement outwards of the great toe is present, but not enough to cause pain. It is by this latter symptom that the surgeon's attention is called to the part, which is seen to be inflamed and prominent. In old-standing cases the altered direction of the great toe, the presence of a fluctuating swelling, and of corns over the inner side of the head of the metatarsal bone, suffice for the diagnosis (Fig. 20).

Occasionally the affection is mistaken for gout, but a little care should serve to distinguish an inflamed bunion from this affection. As the great toe is being displaced it rides over the second toe, and depresses its second and third phalanx so that a hammer-toe complicates the deformity. In cases

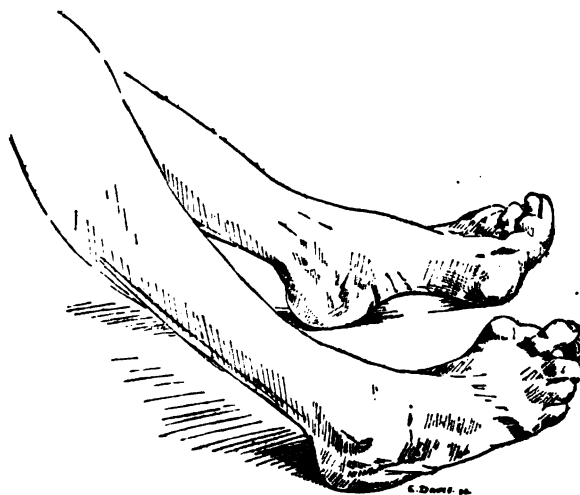


Fig. 19.—A severe case of Bunion.

an important part in maintaining and increasing the deformity; the skin on the inside of the foot and beneath the ball of the great toe is in early cases thickened, reddened, and later becomes much inflamed. Beneath it a false bursa or bunion forms. It is sometimes multilocular, and is very liable to inflammation and suppuration. From these may arise cellulitis. When pus makes its way into the joint disorganisation occurs.

Corns* situated in and on the bunion are extremely painful, and often of large size. In one patient, from whose foot I dissected out a large and painful bunion with a corn on top of it, I found on subsequent section of the part removed that the corn extended through the whole thickness of the bunion—nearly an inch.

of hallux valgus or bunion, flat-foot is often present as well. Women suffer more frequently than men—three times as often—both feet are often simultaneously affected, though not in the same degree.

Treatment.—Prophylactic.—Pointed boots must be absolutely forbidden. It is not of much avail to discuss with the patient whether the boots he is wearing are pointed or not. The outline of the sole of the boot he should wear must be given to him, and he should be directed to go to a rational bootmaker who will carry out directions. The sole of the boot should be as broad as the sole of the foot when it is placed upon the ground and the weight of the body is being borne upon it. If any displacement exist, the inner side of the upper leather should be blocked out, so as to give ample

room to the great toe and to the prominence of the metatarso-phalangeal joint. In all cases it is better to direct a bootmaker to make a 'last,' and then for it to be submitted to the surgeon to see if it fulfil the proper requirements.

Curative treatment consists in slight cases in wearing boots on the lines just suggested, the

well in practice. Pulling inwards the great toe night and morning, if persevered with, bring it into better position.

Various simple arrangements are made, such as a leather cap or thimble fitting over the great toe, and secured by a tape which passes along the inner border of the foot and around the heel, and ends in

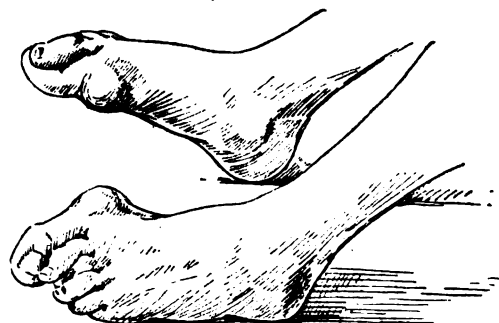


Fig. 20.—A second view of the same feet as in Fig. 19.



Fig. 21.—The appearance of the feet seen in Figs. 19 and 20 after removal of the heads of the metatarsal bones.

application of cold and soothing lotions to the inflamed and thickened skin, and the use of some arrangement whereby the great toe is kept away from the second toe. In early stages nothing answers so well as the divided or digitated sock with a separate 'stall' for the great toe. Some advise the use of a post between the first and second toes, but many do not find this answer

an elastic insertion, which is fastened to the outer border of the foot.

Bunion springs are also made, but they are somewhat difficult of application and not always efficient. When the patient is unable to afford a special apparatus, a wedge-shaped pad of lint fixed between the toes is of service, and the first toe may be further separated from the second by drawing it

away by strapping secured round it and passing back towards the heel. Or a splint of rubber or pasteboard may be fixed to the inner margin of the foot and toe, thus pulling the latter inwards.

When the deformity is severe, the bursa large and painful, and subject to recurring attacks of inflammation, operative procedures are necessary, and only those which involve bone are of any permanent value.

On account of the altered direction of the great toe, the tendon of the extensor proprius pollicis is displaced outwards, and perpetuates the deformity. The necessary preliminary to any operation on the bones is, therefore, division of the extensor proprius pollicis tendon.

Now, the operations on the bones are three in number, namely: 1. Cutting down upon the joint, removing the bursa by the way, and chiselling off the prominences round the head of the metatarsal bone and base of the first phalanx. This gives fairly successful results in cases which are not very severe. But I sometimes find that there is occasionally not so free movement at the joint afterwards as there should be, although I have never found any cases that I have operated to be followed by ankylosis. I am therefore more inclined to recommend the next procedure.

2. Removal of the head of the metatarsal bone. This answers extremely well, the affection is cured, and the patient has extremely good movement and can walk about as well as ever (Fig. 21).

In still more severe cases, where the base of the first phalanx is enlarged, I adopt a third procedure, viz. removal of the base of the first phalanx in addition to the head of the metatarsal bone. About three weeks after the operation a false joint forms between the metatarsal bone and the phalanx, and the result is excellent. For cases of moderate severity, then, which do not yield to ordinary simple measures, the best form of operation is excision of the head of the metatarsal bone. It is quite safe, and I have performed it in a number of cases with complete relief to the patients.

Hallux Varus, or Pigeon-toe.

This deformity is met with frequently in congenital equino-varus, and after the treatment of that affection it may remain as an obstinate feature of the deformity. It varies in extent, and the angle of inward displacement may be 45° or more.

The treatment consists of manipulation and the use of a light splint to press the toe outwards.

Hallux Rigidus.

This affection is also known by the names of Hallux Dolorosus, Hallux Flexus, and Painful Great Toe.

As to the exact description of this affection various accounts are given. For instance, in some cases the toe is held quite rigid and cannot be extended without extreme pain, although it may be flexed without much trouble. The joint, however, is always held rigid, and, as before stated, any attempt at movement causes very considerable pain. In other cases the deformity consists of a forced flexion of from 30° to 60° of the proximal phalanx of the great toe, and some extension of the second phalanx while the toe is held rigidly in that position. Mayo Collier gives the following account of the affection, founded on nine cases, seven males and two females: "the affected foot presents a peculiarly characteristic appearance. In the first place it is a long foot; it is an abnormally long foot for the size of its owner. Next the foot is nearly always cold, damp, and blue. The distortion of the foot is characteristic and peculiar, and is due to the fact that any pressure between the head of the metatarsal bone and the sesamoid bones on the tendons of the short flexor cannot be tolerated. The metatarsal bone is flexed on the tarsus, and is adducted to the mid-line from its fellows. With this the proximal phalanx is slightly flexed. . . . The head of the metatarsal bone appears through the skin to be enlarged, and there is found sometimes a lipping of the cartilage of this bone at its under and lower aspect in the neighbourhood of the sesamoid cartilages. In early cases pain is not usually complained of until the end of the day, and then mostly after long standing or much walking, but as the disease progresses the pain is continuous. The joint is never red, painful, or tender to the touch, except on manipulation. . . . Flexion of the joint is generally readily permitted, but any attempt at extension elicits opposition and evidence of acute pain on the part of the patient." It seems to me that the discrepancy as to the position of the parts is explicable on the supposition that the cases recorded are various stages of one affection.

The disease is more commonly seen in males

than in females, and in young males under twenty years of age. It is generally associated with flat-foot; and, according to Cotterell, its origin is to be sought in a combination of flat feet and short boots. The great toe is thus cramped, the proximal phalanx becomes flexed, and the head of the metacarpal bone depressed. Pressure then follows between the last-named structure and the sesamoid bones.

As to the anatomy of the affection, the plantar fibres of the lateral ligament and the tendinous tissue connecting the sesamoid bones have been found to be contracted, so that extension of the toe is limited. Mayo Collier found caries with marked absorption from pressure existing on the under aspect of the head of the metatarsal bone, at the points of contact with the sesamoid bones. The cartilage was almost completely worn off the points of contact, leaving the subjacent bone bare and congested. There was also some lipping of the adjacent margin of cartilage; and at spots, invasions of granulations passing in from the synovial membrane. The cartilage of the head of the bone, as well as of the proximal phalanx, was healthy.

My own opinion is that the locking is usually due to osteophytic outgrowth from the base of the first phalanx and around the head of the first metatarsal bone; very frequently these exist in the neighbourhood of the sesamoid bones, and may explain the caries which Collier found in that situation.

The treatment is to remove the causes, viz. flat-foot and too short boots. In the majority of cases this is successful. But in inveterate cases I have always excised the head of the first metatarsal bone, and with success.

Hammer-toe.

This deformity is usually one affecting the second toe, and consists of dorsi-flexion of the first phalanx, with plantar flexion of the second, and extension of the third phalanx.

1. *Causation*.—In a small proportion of cases a distinctly congenital origin can be shown. In these instances the second toe is usually affected, and in both feet. In the same patient congenital contraction of the fingers, especially of the fourth and fifth may be seen.

2. *Heredity*.—Of all the deformities to which

the foot is liable, this is the one in which heredity is most marked. Mr. William Anderson has traced this feature in at least a fourth of the cases which have come under his notice, and in the lectures on the contraction of the fingers and toes he alludes to an instance in which the deformity had occurred in four generations. I have met with two examples in private practice of its perpetuation through three generations.

3. *Acquired causes*.—In many people the second toe is longer than the first, and by some this is believed to be a normal state. Whether the first or second toe be longer it matters little if tight and pointed boots are worn, so far as the production of hammer-toe is concerned. In either case, the great toe not having sufficient room is subluxated outwards, and the other toes become clawed. As the displacement of the great toe persists it rides over the second toe, and the second and third phalanges of the latter being maintained in constant plantar flexion, adaptive shortening of the long tendons and the lateral and glenoid ligaments follows, with hyper-extension of the first phalanx (Figs. 22 and 23).

The appearances of hammer-toe.—On the dorsal aspect of the first inter-phalangeal joint a painful corn is frequently present. Beneath this is a bursa which from time to time inflames and suppurates. On the under surface of the joint a deep groove is noticeable. The skin is contracted, and at the bottom of the groove the long flexor tendon can be felt. The first phalanx is in a state of extreme dorsal flexion, so that the head of the metatarsal bone is uncovered below to about half its extent. The second phalanx is always plantar flexed, while the third may be either plantar flexed, dorsi-flexed, or in a line with the second. As a result of the squeezing of the tip of the affected toe downwards, it is often broad and flat, hence the term "hammer-toe Fig. 24").

The second toe in the majority of cases is affected, and in both feet, but to a variable degree. I am inclined to think that the deformity is more common in women than in men.

Morbid anatomy.—Whatever may be the original cause, the great obstacle to complete reduction is found to exist in the shortened lateral ligaments. The flexor and extensor tendons are undoubtedly contracted, but division of them is not sufficient to remove the deformity. The lateral

ligaments must be severed. As a result of the displacement, neither the lower part of the head of the metatarsal bone nor the upper half of the base of the first phalanx is covered, and the cartilage of the uncovered portion of bone is thin and atrophied. On account of inflammation extending from the bursa, ankylosis between the first and second phalanx has been met with.

Treatment.—In slight cases attention to the

is felt, indicating rupture of the lateral ligaments. If the shortened skin prevent complete restitution of the toe, the skin may be divided by a V-shaped incision. But in most cases it is far better to divide the lateral ligaments and the flexor tendons in the following way, and I have treated a very large number of cases of the following kinds, with good results in every instance.

The operation is done as far as possible subcu-



Fig. 22.—A case of Hammer-toe.

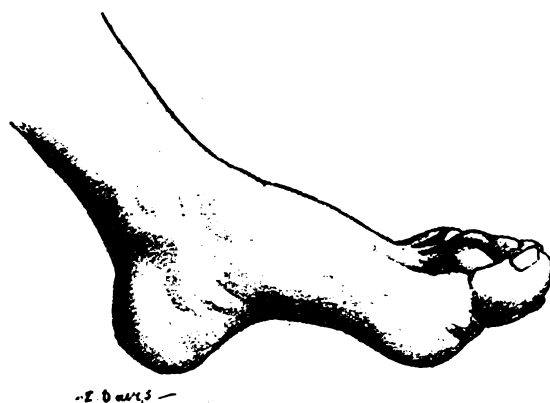


Fig. 23.—Another view of the same foot as in Fig. 22.

boots, and the various measures for remedying the outward displacement of the great toe, detailed previously, are of value, especially if care be taken to straighten the affected toe several times night and morning. By fixing at night a malleable iron splint, suitably bent, to the sole and the affected toe, the condition will often be improved.

Operative.—In cases of slight degree, forcible reposition with the fingers under an anæsthetic is sometimes successful. A distinct snap or crack

taneously, and is performed under aseptic precautions. The assistant holds aside the first and third toes, and the surgeon, steadying the second toe with his left hand, enters a fine, narrow-pointed, strong-backed knife at the mid-point of the groove on the under aspect of the first inter-phalangeal joint, and passing the knife upwards beneath the skin and avoiding the digital arteries and nerves, the edge of the knife is turned towards the bone, and the lateral ligaments are severed. By con-

tinuing the division on the under aspect of the joint, the long tendon and the glenoid ligament are divided. Without removing the knife from the skin puncture, it is passed down the bone through the remaining lateral ligament. After free division

Should the toe, however, not come into good position, the puncture on the under surface may be enlarged to a transverse incision; then the head of the first phalanx is protruded and removed by bone forceps.



Fig. 24.—A third view of the same foot as in Fig. 22, and showing the first and third toes meeting over the second.



Fig. 25.—The same foot as in Figs. 22, 23, 24, showing the hammer-toe cured by division of the flexor tendon and the lateral ligaments.

of the ligaments and tendons the toe can be brought into good position. If not, a little force suffices to rupture any of the ligamentous fibres which may have escaped division. In some cases it is as well to sever the contracted extensor tendon (Fig. 25).

After the operation and dressing, the toe is placed in the corrected position in a malleable iron splint until the wound is healed. A small T-splint is then worn for some time until the toe shows no disposition to return to its former state.

Complete excision of the joint by a lateral incision is practised, and cures the patient ; saving, too, a considerable amount of time in treatment. Amputation is rendered entirely unnecessary if the foregoing plans be efficiently carried out ; and, indeed, it is, in my opinion, an unnecessary mutilation.

CLINICAL CASES

FROM THE

Belgrave Hospital for Children,

February 11th, 1897.

Under the care of Dr. CAUTLEY,

Physician to the Hospital; Assistant Physician to the Metropolitan Hospital.

Tubercular Meningitis. General Tuberculosis.

THE first case is that of a boy *æt.* 6½ years, who was admitted on February 8th with symptoms of meningitis. He is the eldest of five living children, and two others have died from tubercular meningitis. Both parents and grandparents are healthy, and there is no history of phthisis. The father has had syphilis.

Two years ago the boy was in this hospital for a week with bronchitis. Three months ago he had some slight illness for a week, but after that nothing was noticed until January 29th, when he was seized with headache and abdominal pain. From that date up to February 4th the headache persisted, the bowels were confined, and vomiting occurred after meals.

On February 4th he attended as an out-patient, and presented the early signs of meningitis, namely, irritability, headache, irregularity of the pulse, vomiting, and constipation. Two days later he appeared somewhat better. From lack of room he could not be admitted until February 8th, on which day he was decidedly worse. He had no optic neuritis at this time, but on the next day the discs were rather blurred.

He has now been in three days, and is rapidly going downhill. The flushed cheeks, *tâche cérébrale*, retracted abdomen, and comatose condition are very characteristic. There is no squint. The pupils are dilated from the effects of atropine,

and the optic neuritis is well marked. The neck is a little rigid, though the head is not retracted. The pulse is frequent, 144, and small. Some slight convulsive movements of the limbs were noticed two days ago, but none since. He now lies on his back in a comatose condition, passes everything under him, and has to be fed by a tube. The temperature has never been above 101° F.

At the right apex there is a little flattening, with slight impairment of resonance and harsh breath-sounds. Over the manubrium the note is impaired, and the breath-sounds are tracheal in character. A few crepitations are heard at the bases behind. The boy is poorly nourished, and presents no further signs of disease.

The course of the case may be summed up as follows. An attack of bronchitis, possibly tubercular in origin, took place two years ago, and induced enlargement of the bronchial and mediastinal glands. Later on the glands became caseous, and finally general dissemination took place. The physical signs indicate old mischief at the right apex and enlargement of glands in the anterior mediastinum.

The case is very acute, and will terminate in death in the course of a few days, although the temperature is low. The rapidity of the pulse is a bad sign. The question of drainage might be raised. This case is, however, unsuitable for such treatment, by reason of the rapid progress and the evidence of disease in the chest. There is no reliable evidence of excessive fluid in the ventricles of the brain.

Postscript.—The boy died rather suddenly two days later. On post-mortem examination slight basal meningitis was found, no excess of fluid in the ventricles, and no softening. No definite tubercles were discovered in the meninges. Situated at the back of the manubrium, to the right and somewhat in front of the trachea, was a mass of caseous glands, an inch and a half in vertical measurement, and an inch transversely and antero-posteriorly. The gland at the bifurcation of the trachea was also large and caseous ; so, too, many glands in the posterior mediastinum, especially on the right side. The upper lobe of the right lung had undergone a certain amount of fibroid change, but no evidence of old tubercular deposit was found. At the base of the upper lobe there was a considerable deposit of recent grey

and yellow tubercles. At the root of the right lower lobe was a caseous gland, adherent to the lung, and radiating outward from it to the surface of the lung were striæ of grey and yellow tubercles. A similar caseous gland and condition of the left lower lobe were present to a smaller extent. Miliary tubercles were also found on the surface and in the substance of the spleen. The other organs were healthy.

Tubercular Peritonitis.

The next case also illustrates the wide-spread mischief produced by the tubercle bacillus. It is that of a boy æt. 7, with a family and past history that contain no evidence of tubercular disease.

For a year he has been ailing. For the last ten weeks he has been disinclined to play, and has complained of abdominal pain and headache. Loss of flesh has been noticed for three weeks. The bowels have been rather confined.

The boy is anæmic, looks delicate, but is fairly nourished. On physical examination nothing further abnormal is found until we come to the abdomen. It is full, tense, resistant to examination, and somewhat dull on percussion in the flanks and below the level of the umbilicus. No thrill can be felt. The girth at the level of the umbilicus is 22½ inches.

He has now been in five days, and nothing further has been made out, except that the temperature is irregular, and at times reaches 102° F. His appetite is fairly good, his food is well digested, and he has no pain. Possibly the mesenteric glands are enlarged, but owing to the rigidity of the recti this cannot be ascertained. The treatment consists of rest in bed, local applications of ext. belladonna and glycerine, a mixture of cod-liver oil, malt and syr. fer. phosph. co. internally, and a nutritious, easily digestible diet, with a couple of ounces of port wine.

The diagnosis is tubercular peritonitis. The prognosis is fairly good, but must be guarded. Many of these cases recover completely.

Capillary Bronchitis. Enlargement of the Liver and Spleen.

This little girl looks the picture of health. She was admitted a week ago for capillary bronchitis, enlargement of the liver and spleen, and a small amount of ascites. The last cleared up after

two days in bed. The lungs still show evidence of capillary bronchitis, especially at the bases behind. The abdomen is full. The superficial veins over the upper part and over the lower ribs are unnaturally prominent. The liver reaches almost to the level of the umbilicus, and has a firm rounded edge. The spleen extends two inches below the costal arch, and its edge is firm and rounded.

The interest of the case lies in the diagnosis of the cause of these enlargements. The family history is not good. Consumption is present in the father's family. The mother has had two miscarriages and eight living children, of whom three are dead. One of these had a cleft palate. This history suggests syphilitic infection.

The child herself shows no sign of inherited syphilis. She suffered from rickets in infancy.

The probable cause of the enlargement is either congenital syphilis or rickets. The evidence of the former is by no means conclusive. On the other hand, the child does not appear to have suffered much from rachitis, and we see many cases of rachitic infants without such evident enlargement. The increased size of the superficial veins and the slight ascites indicate some obstruction to the circulation through the liver. The prognosis is good. Many infants and young children come under notice with similar conditions of the liver and spleen, and are generally discharged in good health, suffering no inconvenience from the local condition.

Lobar Pneumonia.

This little girl has just been admitted. She is a year and eight months old, and was brought up by hand on diluted cow's milk. Her mother died at the age of twenty-three of phthisis; her father is healthy, and so is an elder child, aged three.

Five days ago she took ill, was feverish, and "lay about a great deal." Two days ago, not being any better, she was brought to the Out-patient Department. She was flushed, and had a temperature of 104° F., but no abnormal physical signs were found in her chest.

To-day the left cheek is markedly flushed; the respirations are very frequent, over 70 per minute, and the pulse 120. The tongue is moist and thickly furred on the dorsum. On examination of the chest the skin is noticed to be "burning"

hot. At the right base in front there are signs of consolidation below the level of the nipple; namely, fine crepitations, bronchial breathing, and dulness. A patch of consolidation is found on the right side behind, just below the angle of the scapula; here the note is much impaired, and the bronchial breathing is intense. Over the remainder of the right lower lobe the note is impaired, and there are a few crepitations. The temperature is 103° F.

No doubt the diagnosis is pneumonia of the right lower lobe with partial consolidation. The localisation of the dulness might suggest that it is a case of lobular pneumonia, a disease so common in children during the first few years of life; and the mode of onset, there being no history of vomiting, headache, or convulsion, is rather in favour of the latter diagnosis. On the other hand, there is no evidence of a catarrhal condition of the lungs, and there is evidence that the whole of the lower lobe is becoming involved. In all probability by to-morrow complete consolidation will have taken place.

The treatment will consist of small doses of vin. ipecac., ammon. carb., and tinct. digitalis, flavoured with aq. caru. For the present the diet will consist of milk. Half a drachm of brandy will be given every three hours. It is not my custom to give brandy in every case of pneumonia, but for the present patient it is indicated by the severity of the symptoms and the weakness of the first sound of the heart. Oxygen inhalations will be used if the colour becomes bad, or the child seems to be failing.

The prognosis is good, although the child is very seriously ill and under two years of age. The disease appears limited to one lobe, and there is no evidence of any complication. The prognosis must be guarded, as it is quite impossible to estimate the effects on the heart of the toxins produced, and in the present case the condition of the heart suggests that it will require careful watching and stimulation. One favourable indication is that the child takes nourishment readily.

Caseous Bronchial Gland ulcerating into the Œsophagus.

The little girl in this bed has just been admitted. She is eight years of age, and has been ailing for five or six years, being under treatment at various

hospitals and dispensaries. There is no family history of tubercular disease.

For eight months she has suffered from cough. During the last month she has suffered from diarrhoea and vomiting.

She is extremely emaciated, merely skin and bone, and weighs only $30\frac{1}{2}$ lbs. On examination nothing abnormal is found in her chest, save a few moist sounds, certainly not enough to account for the extreme wasting. The abdomen is flaccid and easily examined; no enlarged glands can be felt. There is moderate œdema of the feet and lower two-thirds of the legs. The motions are very offensive, and resemble pea soup in appearance and consistency. The temperature is subnormal.

This child resembles in appearance and symptoms a girl aged five years, who died in here last year from hæmoptysis a few days after admission, and who was found post mortem to have a small ulcer in the œsophagus, through which a broken-down gland at the bifurcation of the trachea was discharging purulent and caseous matter. The abscess cavity also communicated with the bronchus and the lower lobe of the right lung, which was partially solidified and riddled with tubercle. The hæmorrhage had come from the abscess cavity into the œsophagus, and not through the bronchus. The child was very wasted, had suffered from offensive breath, foul-smelling stools, and vomiting.

I suspect a similar condition of affairs to exist here. The prolonged cough may have been caused by reflex irritation due to the enlarged gland, while the diarrhoea and vomiting may have followed on its rupture and discharge into the œsophagus. At present there is no evidence of the lung tissue being involved, as it was in the other case, in which marked dulness and bronchial breathing were found over the right lower lobe.

The prolonged history of delicate health is strongly in favour of some tubercular process. The continued offensive diarrhoea, uncontrollable by drugs, indicates some persisting infection of the alimentary canal. Another possible diagnosis is a simple chronic enteritis.

The prognosis is bad. Possibly the gland might discharge its contents, and then undergo contraction, but such a result is unlikely. Tubercular ulceration of the intestines may also ensue, and may even now be in existence.

Pleurisy with Effusion of Lymph.

Here is a boy *æt.* 4, who has been ailing for four months, and for the last three months has had abdominal pain and diarrhoea. For two weeks he has had a troublesome cough. There is a doubtful family history of consumption, and a past history of childish complaints.

He is a small, thin, pale, delicate child. The heart-sounds are normal, the apex in the fourth space in the nipple line. Behind, the note is impaired over the left lower lobe, especially below the angle of the scapula. The loss of resonance becomes less marked on approaching the spine and on passing outwards into the axilla. Over the dull area the breath-sounds are feeble as compared with the opposite side, but there is no bronchial breathing and no tenderness. No friction is heard.

The diagnosis is pleurisy with exudation of lymph covering part of the left lower lobe. The possibility of localised empyema suggests itself, and in favour of this are the long history of illness, the temperature, and the diarrhoea. On the other hand, the boy hardly seems ill enough; he is not markedly anæmic, and there is no local tenderness, and no displacement of the heart. The diarrhoea may be due to food unsuitable in character for his debilitated health. There is nothing characteristic about the stools, except that they contain undigested food. The temperature is raised, up to 101° F. at times, and is irregular. I expect that the physical signs will clear up slowly, but if not, I shall explore in the course of a week or ten days.

Marasmus.

The last case illustrates some of the methods which can be adopted for the feeding of marasmic infants. Here is a boy who was admitted on December 7th, at the age of four months, and weighing only 5lbs. 1 oz. He is one of thirteen children, seven of which are living. Eight of these children, including this one, have been insured. I am afraid that the mortality among insured infants is considerably higher than among the uninsured. On admission he was extremely emaciated, the face small and sunken, with the aspect of an old man; the skin thin and shrivelled, easily picked up into loose folds. The parietal bones overlapped, and were thin. There was no

evidence of craniotabes. The abdomen was prominent and somewhat rigid; the liver and spleen could not be felt. There was no rash, no scarring about the mouth, nor other sign of congenital syphilis.

The child first came under my care in the Out-patient Department at the age of six weeks, and had been brought to see me fairly regularly since. The mother states that the child has been fed on the breast solely, and has steadily wasted since birth. She is an untruthful, moderately fat woman, *æt.* 39, and ought to have been able to bring up her child on the breast perfectly satisfactorily. Her failure is probably due to irregularity in feeding, and to her habits of life, perhaps assisted by the fact that it is her thirteenth child and insured. That she is not a good mother is shown by the history that she has already lost six of her children. During the attendance of the child as an out-patient the mother did not carry out the instructions given her as to feeding, and the child never made any real progress. It was admitted after an attack of diarrhoea of three days' duration.

On referring to the charts and diet sheets the important points to notice are the weight, diet, frequency of the bowels, and the temperature.

	<i>Weight.</i>	<i>Gain.</i>
Dec. 7th . .	5 lbs. 1 oz.	
Dec. 14th . .	5 lbs. 7 oz.	6 oz.
Dec. 21st . .	6 lbs. 2 oz.	11 oz.
Dec. 28th . .	6 lbs. 4 oz.	2 oz.
Jan. 4th . .	6 lbs. 6 oz.	2 oz.
Jan. 11th . .	6 lbs. 11 oz.	5 oz.
Jan. 18th . .	6 lbs. 12 oz.	1 oz.
Jan. 25th . .	6 lbs. 13 oz.	1 oz.
Feb. 1st . .	6 lbs. 14 oz.	1 oz.
Feb. 8th . .	7 lbs. 3 oz.	5 oz.
Feb. 11th . .	7 lbs. 3 oz.	0 oz.

Thus the total gain amounts to 34 oz. in 68 days, an average of half an ounce a day. The gain in weight has not been at all regular, and, while quite satisfactory during the first few weeks, has been very insufficient since January 11th. The explanation of this may lie in the diet, the temperature, or the condition of the bowels.

At first the diet consisted of a mixture containing about 0.5 per cent. fat, 0.75 per cent. proteid, and 5.0 per cent. sugar. It was prepared by mixing milk 1½ oz., whey 5½ oz., water 6½ oz.,

sugar $1\frac{1}{2}$ oz., lime water $1\frac{1}{2}$ oz. Two ounces of this were given every two hours, night and day. The child took the food well, digested it, and had no diarrhœa.

On December 14th the percentage of fat and proteid was a little increased. Two and a half ounces were given every two and a half hours of a mixture of cream 1 oz., whey 16 oz., sugar 1 dr., lime water 1 oz. This also was well digested and assimilated. There was a gain of 11 oz. in the next week.

From December 19th to 30th the temperature was somewhat raised and irregular, once reaching 100° F.; the bowels were rather relaxed, and the child had attacks of opisthotonos. There was a small loss of weight. These attacks of opisthotonos have continued on and off with varying severity up to the present time.

During the next week the temperature kept at a lower level, the bowels acted less frequently, and the child again gained weight.

Another ounce of cream was added to the mixture on January 7th; after this the rate of gain became less, the temperature was higher and more irregular, and the child did not improve.

On January 21st the diet was altered to one of cream 2 drs., milk 1 oz., water $1\frac{1}{2}$ oz., sugar 1 dr., lime water 2 drs., seven feeds being given at intervals of three hours. On the 28th this was diminished in amount; cream 1 dr., milk 6 drs., water 1 oz., sugar 1 dr., lime water 1 dr. being given for each feed, and nine feeds in the twenty-four hours.

On February 1st a teaspoonful of raw meat juice was added to each feed.

Reference to the weight shows that the child does best on a diet containing a very low percentage of fat; when this food is increased the gain of weight is less and the bowels act too often. The proteid is best digested when given in a very assimilable form, as lactalbumen in whey, and as albumen in raw meat juice.

It is noticeable that the rate of gain is much affected by the height of the temperature. The temperature and the opisthotonos, with which is also some rigidity of the neck muscles, suggest that there may be some organic mischief, such as basal meningitis. Such a condition would readily account for the slow progress made lately, but I am disinclined to accept this view for the

reason that though the child is not gaining as it should do, it does not lose weight. I think the explanation of this is that we have not yet found a suitable diet for it, and that the opisthotonos is due to reflex irritation from the intestinal tract.

I propose to try a diet of egg-albumen and sugar, without the addition of fat, except small doses of cod-liver oil. For this mixture I shall order the white of one egg to be mixed with eight ounces of water and half an ounce of sugar. Two to three ounces of this will be given every two and a half hours. It is a very nutritious, digestible, easily assimilable food, and readily taken. I have known many infants do remarkably well on it.

It is an important fact to bear in mind, and well illustrated in the present instance, that marasmic infants do not digest fat well, and will often do better without it. This, I think, accounts for the success sometimes obtained by the use of condensed milk in these cases when given as a temporary food.

NOTES.

Pneumococcus Peritonitis in Children.—

It commences with violent pain in the abdomen, fever, vomiting, and diarrhœa. In a short while this general commotion subsides, and the fever assumes the characteristics of a suppuration fever, while a dull, thick tumefaction appears in the iliac fossa above the pubes, in some cases with fluctuation. This local lesion is significant alone, but it assumes almost pathognomonic importance when it is accompanied by the special sign which Professor Brun has found mentioned in nine of the fourteen cases on record, the swelling and inflamed appearance or fistulization of the umbilicus. In these fourteen cases, three died; one recovered after spontaneous evacuation through the umbilicus and into the vagina. The remainder recovered after laparotomy. His own experience has been four recoveries in five cases. The operation required is very simple, scarcely more than opening an abscess. In the fourteen cases, all under fifteen, the diagnosis was confirmed by bacteriologic investigation. He suggests that the latter fact may elucidate the methods of peritoneal infection, as in every case where the lesions were clearly defined they were located in the lower part of the serosa.—*Presse Méd.*, February 27th.

THE CLINICAL JOURNAL.

WEDNESDAY, MAY 26, 1897.

CLEFT PALATE.

BY

W. ARBUTHNOT LANE, M.S.

GENTLEMEN,—I propose to take as the subject of this address the conditions of imperfect development of the septum between the mouth and naso-pharynx, comprised under the term cleft palate.

In order to place their treatment on a scientific basis, I will first call your attention to the physiology of the nose, which practically means a consideration of the mechanical factors that determine the form of the nasal cavities and of the upper part of the pharynx, described together as the "naso-pharynx." We shall then be in a position to formulate general principles on which our methods of treatment may be most advantageously based.

The chief function of the naso-pharynx is to transmit air into the lungs, in order that the sensitive surface of its mucous covering may become affected by substances contained in it. In other words, this part of the body is constructed essentially for the purpose of smell, and in the several groups of animals the degree of its development varies with the importance of the sense of smell to them.

The upper portion of the nasal cavities is used for the ramifications of the olfactory nerve, and the current of air is distributed especially over this area by the act of sniffing. During the normal process of respiration the mouth is kept shut, the upper lip covering the upper incisor teeth entirely, and projecting below them. During normal respiration the breathing air passes chiefly through the lower meatus, while there is hardly any current in the upper part of the space. It is therefore obvious that anything interfering sufficiently with the calibre of the lower part of the nasal cavities impairs their capacity of transmitting air to an extent to make it necessary to keep the mouth open while breathing. When this

is done, air enters so freely in through the larger and more direct channel formed by the mouth, that it ceases to pass through the nose either during inspiration or expiration, the most important function of the part is lost, and the forces which act upon it normally and develop it, are in abeyance. Even after air has ceased to pass through the nose in inspiration, it does so during the expiratory process of vocalisation, though in a diminishing degree. Therefore the so-called nasal intonation does not develop as early as one would imagine in association with much loss of nasal inspiration. You see at once from an ex-

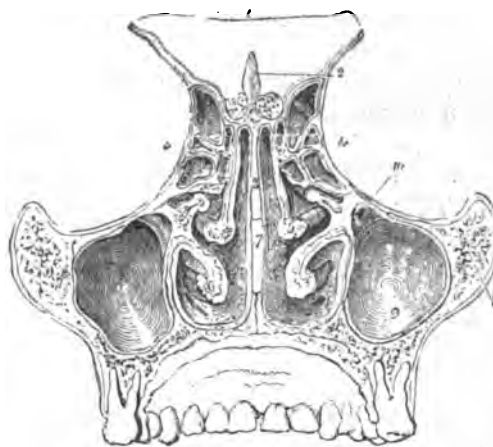


Fig. 1.—Represents a vertical transverse section through the nasal cavities and adjacent structures.

amination of Fig. 1, which represents a vertical transverse section through the nasal cavities and surrounding structures, that the calibre of the lower part of the nasal cavities, or of their respiratory section, if I may so describe it, is enormously encroached upon by any ascent of the hard palate above the normal level, and that the air-transmitting capacity of the naso-pharynx varies inversely as the height of the palate. Also that the calibre of the entire nasal space is very much affected by the slightest variation in the interval between the septum and the structures forming the outer wall of the space.

You all know how exceedingly common are

these cases of imperfect development of the nasal cavity. It has probably often struck you that if we exclude those diseases of the nose, ear, larynx, and even lungs, which are directly or indirectly consequent upon some imperfect development of the naso-pharynx, a not unimportant section of the consideration and treatment of the diseases of these parts would have to be deleted from works on these subjects.

It behoves us, therefore, to consider very carefully the factors which are responsible for such imperfect development, in order that we may save our patients from innumerable complications, some of which may not at first sight seem to depend directly upon it.

I believe that such undeveloped conditions result solely from the fact that for some reason or another the mechanical factors that affect the development of the naso-pharynx are in abeyance, and that in consequence this space does not enlarge as it should.

Of course, we must bear in mind that some children start with, or in other words inherit, a degree of development which is less than the normal. An interesting and ready means is afforded, by the study of such imperfectly developed conditions, of determining how quickly an acquired deformity in the parent may arise in the offspring. Personally I believe that the transmission of this particular condition is very direct and rapid.

The absence of this developmental factor is brought about by an infection of the mucous membrane of the nose, of which the most common type is a cold in the head. The inflammatory swelling of the mucous membrane and the presence of mucus interfere sufficiently with the entry of air during inspiration to make it necessary to breathe through the mouth. This at once deprives the naso-pharynx of the presence of the mechanical factor upon which it is dependent for its growth, namely, the pressure of the air upon its walls; and in consequence it ceases to increase in size. The interval between the septum and the outer wall of the space remains unchanged, and the septum between the mouth and nose does not descend, the arch of the palate remaining abnormally high.

The infective process in the mucous membrane of the nose sets up a corresponding process in the

lymphatic mass in the pharynx spoken of as the pharyngeal tonsil, and later in the lymphatic glands in the neck, which receive part of their supply from these structures. The pharyngeal tonsils are also frequently affected, and the larynx, trachea, and bronchi may be also attacked.

The enlargement of the pharyngeal lymphatic tissue interferes with the normal functions of the middle ear, with consequences you are well aware of; while tubercular organisms readily find a suitable nidus in the inflamed cervical lymphatic glands, and produce more or less disastrous results.

To the condition of nasal obstruction the term "adenoids" has been applied. It has become a household word, and is deservedly regarded by parents generally with very considerable dread.

Unfortunately for their patients, surgeons under the influence of the suggestion of Wilhelm Meyer have considered that the secondary infection of the so-called pharyngeal tonsil is the primary cause of the obstruction of the naso-pharynx, and they have hoped to cure their patients by cutting away a varying proportion of this substance. It is quite apparent that the removal of this lymphatic tissue cannot alone restore to the nose the mechanical factor which is the active agent in its development; though associated with forcible ventilation of the space, such a procedure may occasionally be of service. The children who have not got the vigour and energy to get rid of the primary nasal infection or cold in the head by forcibly expelling the mucus, and drawing air through the space, are very much alike in certain particulars. They are of low vitality, having little energy to expend, assuming habitually attitudes of rest, and avoiding those of activity. The amount of air they change during respiration is very small, and results almost, if not entirely, from the action of the diaphragm, the chest being retained in the position of rest or of expiration. If a tape be passed round the chest at the level of the nipple it will be found to vary to a minute extent, if at all, during natural respiration. Fig. 2 shows such a condition fixed as the "symmetrical resting posture of the trunk," or, as it is often described, "dorsal excurvation." The assumption of the "asymmetrical resting posture" results after a time in its fixation, to which the popular but singularly unmeaning and unscientific terms "lateral curvature" and "scoliosis" are usually applied.

The "fixation of the resting position of the foot and knee" result in the deformities spoken of as "flat-foot" and "knock-knee."

Such children being supplied with a minimum amount of oxygen, and overloaded with pulmonary and other foul products, have no energy to expend in spontaneously and forcibly ventilating the nasopharynx, but at once fall back on the more ready mode of obtaining air through the open mouth, with the results with which we are all too familiar.

It is curious that medical men devote so little attention to the manner in which people perform



Fig. 2.—Shows a typical example of the fixation of the symmetrical posture of rest of the trunk.

the function of respiration, since it is by far the most important of any to the well-being of the individual. By means of it alone are the tissues of the body supplied with oxygen; and in proportion as the supply provided is good or bad, so are the functions of the several tissues and organs performed in a satisfactory or unsatisfactory manner.

The attention of the profession has been fixed, and medical men have educated their patients to fix theirs largely upon the rate at which the products of digestion pass along the intestinal tract, and it is generally assumed that there should be a daily evacuation. I hardly ever find that any attention has been bestowed upon the manner and character

of the respiratory process, though it is of infinitely greater importance to the patient; indeed, constipation frequently and readily results as a natural sequence both directly and indirectly from imperfect oxygenation.

Too often one finds that the patient who performs no thoracic respiration whatever, has the spine fixed in a position of extension by a steel support, or by some form of brace; while at other times exercises are ordered which rapidly exhaust the imperfect capital of oxygen without adding to it appreciably.

This all results from an imperfect knowledge of the mechanics of the respiratory process, and especially of the important part which the spinal column takes in it.

The physiologist has misled the profession chiefly by his attempts at illustrating the respiratory process by means of a mechanism in which the spinal column is represented as a rigid immovable rod. As is too often the case, the generally received teaching is absolutely false, since the variations in the spinal column are infinitely more conspicuous than those of the rest of the wall of the thorax.

I would, therefore, urge that the mode in which the organism is supplied with oxygen should receive at least as much attention as the rate at which the sewage products are evacuated from the body. If this is done, and done well, adenoids, the several varieties of resting deformities, and many other conditions directly and indirectly dependent upon imperfect aëration will disappear.

I do not propose to consider the subject of post-nasal adenoids, as they are called, beyond stating that operative procedures are, in my opinion and experience, quite unnecessary, and that systematic ventilation of the lungs and naso-pharynx furnishes us with a means not only of applying to the naso-pharynx such force as is exerted by air being driven forcibly through it, but by oxygenating the blood more fully, and removing more thoroughly its carbonic acid, &c., the several structures of the body are better nourished, and perform their functions in a normal manner.

As to how this is best done must vary largely with the intelligence of parents, their perseverance, and the time and means they have at their disposal.

In out-patient practice, in which from the circumstances of the parents it can rarely be carried out in

the same effectual manner that is possible in private practice, where the patient is under the immediate control of the medical attendant, and his instructions are strictly enforced, I give the mother a printed form containing the following words, "Put the child on its back three times a day for half an hour at a time, and make it breathe in and out as deeply as possible through the nose, the mouth being kept shut," and endeavour to interest them in its application.

Among the better class of mothers, such as the wives of policemen and mechanics, you will frequently obtain results as good as those in private practice; but the poorer portion of the community have not the time, nor will they often devote their energy to follow these instructions.

The surgeon can readily determine whether his treatment is carried out efficiently or not by making the parents keep a diary in which they must enter the daily differences between the measurement of the chest in extreme inspiration and extreme expiration. To put down the measurements of these instead of the differences would be of little service, since the former vary within wide limits with the exact level of the tape.

Well, gentlemen, I hope I have proved to your satisfaction that the pressure exerted by air, as it is driven backwards and forwards through the constricted channel bounded laterally by the upper jaws, is the only mechanical factor which determines the form of the naso-pharynx, and of the structures which are to a large extent dependent upon it for their perfect development.

Now returning to cleft palate, which forms the subject of this paper, you recognise that while the nasal cavities are in communication with the mouth the mechanical factor upon which they depend for their development is in abeyance, and therefore they do not develop. In consequence the sides of the alveolar arch become approximated, as do the edges of the cleft, and the portions of the roof on either side of the cleft become more vertical and extensive. I will call your attention particularly to this, since you will find that many surgeons use as an argument in favour of delaying the operation that the changes in the roof of the mouth which I have indicated take place in time, and render surgical interference more simple. In other words, the less developed the nose, the more easy the operation for the closure of a deficiency in its floor.

Since the calibre of the nasal cavities bears an inverse proportion to the height of the palate, and as the lower part of the nasal fossæ is that through which air is chiefly transmitted, this is especially encroached upon by any abnormal increase in the height of the palate. Being aware of the mechanics of the naso-pharynx, you will recognise the immense importance of separating the mouth from the nose as early in life as possible, so that the pressure exerted by the air as it passes through the former can be brought to bear upon the walls of the space, so that the nasal cavities and the adjacent bones shall be influenced by normal developmental factors.

In order to recall to your minds the practice followed by surgeons of the present day, I will quote a few lines on the treatment of cleft palate from Treves' 'System of Surgery,' which is, I think, the most recent English text-book. "In the infant the cleft is by almost all surgeons rightly deemed inoperable. In the child, after the age of two years, the chief importance of the malformation is the defect of speech which is occasioned. It is well, therefore, to be prepared for the fact that the closure of the cleft in no way remedies the defect of articulation. Of prime importance is the question, at what age should the operation be performed? It may, I think, be laid down that whilst it is never wise to operate on a child under three years of age, the time of election is from this age up to six years." These definite statements, which are picked out from the text, we may regard as representing generally accepted views, and as open to our criticism. The author does not appear to adduce any argument in support of the first statement, viz. that surgeons are acting *wisely* in avoiding operations in infants; therefore we may satisfy ourselves by merely asserting that in our opinion the statement is false, and that the reverse is true. The second statement is, I fancy, partly correct. Some adult patients will, however, complain more of the discomfort they experience through food getting into the nose, than from the consciousness of the fact that their voice differs from the normal. As to which you consider the chief importance of the deformity depends upon whether you regard it from the point of view of physical discomfort to the sufferers, or of depreciation of them in the opinion of their fellow-creatures, since the peculiar intonation, *per se*, causes no discomfort, and is often not

recognised by the speaker. However, from the point of view of the person not the patient we may readily admit the truth of the statement.

Now as to the statement that "it is well further to be prepared for the fact that the closure of the cleft in no way remedies the defective articulation," this I am prepared to deny if the operation is performed during the period of growth. The reason that the patient's intonation is peculiar, or, as it is usually called, nasal, is that the outgoing air does not pass through the nose, and when the cleft is closed at the usual time, there is in the extremely undeveloped nose very little space through which air can be transmitted, and as far as I am aware no effort is made by surgeons to develop this space after operation. This is probably due to the fact that they had not considered the mechanics of this channel. If, however, the space is systematically and forcibly ventilated the calibre of the naso-pharynx is increased, and a considerable proportion of air passes through it, so that the so-called nasal character of the voice is diminished very greatly. Still assuming this statement and the preceding to be absolutely correct, in what light are we to regard the next? "Of primary importance is the question, at what age should the operation be performed? It may, I think, be laid down that whilst it is never wise to operate upon the child under three years of age, the time of election is from this age up to six years." If we allow the second and third statements to be true, as they are approximately so, it is difficult for the ordinary mind to grasp the logical consequence of the conclusion deduced from them. No, gentlemen, the treatment of cleft palate, like the greater part of surgery, has been a matter of creed and tradition, and has not been arrived at in any reasonable manner. In order to treat these cases as efficiently as possible no time whatever should be lost in restoring to the nose its normal physiology, or in other words of giving back to it the mechanical factor which alone determines its development and that of the other structures dependent on it, to a considerable extent for their normal form.

We will pass on to consider the following questions, viz. :

1. What is the best age for operation?
2. What is the best method of performing the operation?

3. How and when any complication such as harelip can be met to the greatest advantage?

1. *Age.*—I find the best age for operative interference is during the fifth week, providing there is no special indication to the contrary, by which I mean bronchitis, diarrhoea, and the results of bad feeding generally. Under such circumstances it is necessary to postpone the operation till the condition of the child is satisfactory.

The advantages of operating at this early period are—

- (a) The child bears the operation very well.
- (b) The child experiences but slight subsequent discomfort, and will take its food with satisfaction within an hour or two of the operation.
- (c) The amount of hæmorrhage is very slight, and is readily controlled.

2. *Method of Operating.*—Though the details of the operation must vary considerably in a small proportion of the cases, in a large number I endeavour to raise a flap of mucous membrane and periosteum from one side, and to fasten it securely beneath the separated margin of the opposite side. This is to a certain extent a modification of a mode of treatment by flaps introduced by Mr. Davies Colley some years ago.

The measures I adopt can be best illustrated by diagrams. Fig. 3 represents a cleft running through the whole of the hard and soft palate. A flap which is attached to the margin of the cleft is turned up from the bone of the hard palate, and is dissected off the soft palate. Great care must be taken not to tear away this flap from the margin of the cleft in the hard palate. The latter part of the operation can be done best with the form of knives shown in Fig. 4. In doing this the descending palatine vessels are exposed as they emerge in a loose periosteal sheath from a single foramen. These can be cut long, and any hæmorrhage readily controlled by a ligature or by torsion. An incision is then made along the opposite free margin of the cleft in the hard palate, and the muco-periosteum is raised from the bone for about a quarter of an inch. The soft palate on the same side is pulled forward, being held in forceps, or a thread may be passed through its extremity. It is split in the same manner indicated in Fig. 5, none of it being removed. Lately I have frequently cut a flap from

the posterior aspect, increasing still further the areas of the opposing raw surfaces. This is done in order to offer a large raw surface which can be brought into accurate apposition with the reflected flap. The reflected flap is first introduced beneath

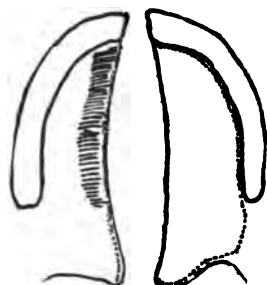


Fig. 3.—Represents a complete cleft of the palate. The dotted lines indicate the incisions, and the shaded area the portion of muco-periosteum elevated from the subjacent bone.

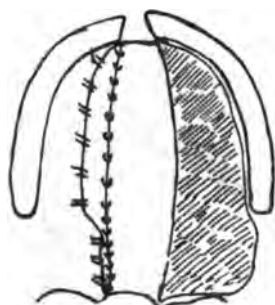


Fig. 3 a.—Represents the flap fixed in position by a double row of sutures.



Fig. 3 b.—Shows in vertical transverse section the line of incision along the margin of the flap.



Fig. 3 c.—Shows the flap raised and fixed in position. It is a transverse section through Fig. 3.

the separated edge of the muco-periosteum of the hard palate, and its margin pinned at intervals by sutures. Then the margin of the flap of the soft palate is similarly attached to the outer limit of the raw surface on the back of the opposite margin by

means of separate sutures. The free part of the elevated border is attached securely to the raw surface of the reflected flap by means of separate or several continuous sutures. In this manner two large raw surfaces well supplied with blood-vessels

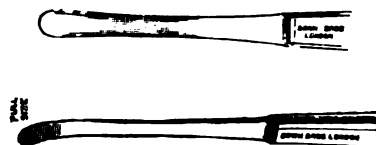


Fig. 4.—Represents straight and curved knives used for splitting the soft palate.

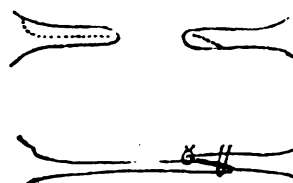


Fig. 5.—Shows manner of treating soft palate.

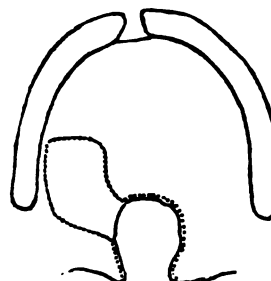


Fig. 6.—Mode of treating residual cleft in soft palate.

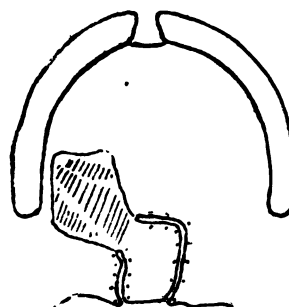


Fig. 6 a.—Mode of treating residual cleft in soft palate.

are retained immovably in accurate apposition, and are placed under the most favorable circumstances for immediate union. It is very unusual for any portion of the flap covering in the cleft in the hard palate to give way, but occasionally the

margins of the soft palate may become apart. To remedy this, after an interval of two or three weeks, a flap should be taken from the side of the hard and soft palate opposite to that utilised on the first occasion, and be retained by the same method in the cleft (Fig. 6). By this means the sutured

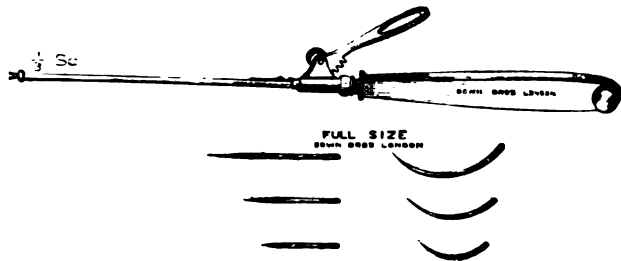


Fig. 7.

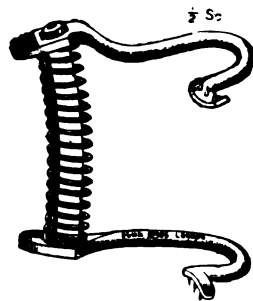


Fig. 8.

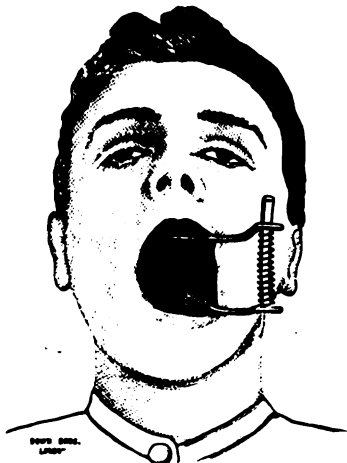


Fig. 9.

surfaces are exposed to a minimum of strain. To render this method of operating in the young infant possible, it was necessary to devise an instrument for the purpose, since those in common use at the time

were inefficient. The needle-holder and needle shown in Fig. 7 were made for me by Messrs. Down Brothers, and meet all possible requirements. The needles are made straight and full curved, and vary in size from three-eighths of an inch upwards. For pinning the flap the straight needles are best suited, while the curved ones are more serviceable in the later stages of the operation. The sutures used are of the finest silk, and employed dry to facilitate threading. The gag I employ is one made for me by the same firm some time ago. It is perfectly self-retaining, owing to the presence of small sharp teeth which bite into the gum behind the molars. It is depicted in Fig. 8, and in position in Fig. 9. It is well to be provided with several sizes of this gag. The gag most generally used is too powerful for the infant only four weeks old. Messrs. Weiss made me a very useful pair of forceps which have terminal teeth, and approximating serrated plates (Fig. 10).

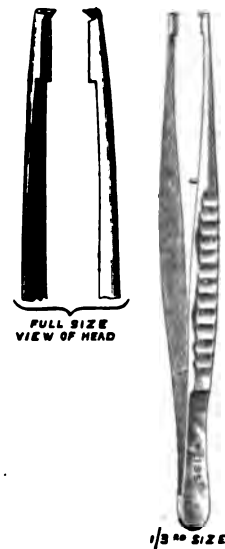


Fig. 10.

By means of the teeth the flap can be held, and with the plates the operator can grasp the needle firmly and readily, and draw it through the flap when necessary. As a rule, the needle is taken up and drawn through the flap by the needle-holder.

Many children with cleft palates are also afflicted with harelip. The latter is, of course, left untouched till the fissure in the palate has been closed, since it affords a larger aperture, and

renders the cleft more get-at-able and the operation consequently very much easier to perform than it would be if the harelip was treated in the first instance. When there is no cleft in the lip and the narrow orifice is a difficulty, I do not hesitate to split the lip, restoring its continuity accurately after the operation on the palate has been completed.

I do not pretend to obtain the same results in the treatment of harelip as are apparently got by some surgeons, if one can place any reliance whatever upon the diagrams they use to illustrate their methods of procedure. These, I fancy, are purely imaginary, since they appear to be able to restore the imperfectly developed lip to its normal form, and to its relationship to the lower lip. My experience is that, excepting in slight cases, the upper lip always remains smaller than normal, and disproportionate in size to the lower. In some few cases I have obviated the relative loss by filling in this gap by a flap obtained from the lower lip, with which it remains continuous till it has united firmly to the upper. I would point out to you the advantage of passing all the sutures through the lip from within outwards, since by so doing you can bring the margins of the cleft into firm and accurate apposition, and are enabled to control the hæmorrhage without scarring the skin surface, as is frequently done when the sutures pass through the skin in the usual manner.



Fig 11.

Besides, they can be left in much longer, as their presence causes no discomfort, and retains the raw surfaces in accurate apposition. The scarring of the lip was a more marked feature when the old-fashioned harelip pins were employed.

There is a form of cleft palate combined with double harelip, in which the premaxilla is attached

to the under surface of the septum of the nose immediately behind its tip (Fig. 11). In such cases I have found the most satisfactory procedure is to close one of the fissures in the lip in order to supply the premaxilla with blood-vessels from a source other than the septum. When this has been effected, the premaxilla can be cut away from the septum, the mucous membrane covering it, and the corresponding surfaces of the jaw removed, and the bone being trimmed and replaced can be wired securely in position. The remaining fissure in the lip can then be closed, but by leaving it open till this period you obtain more room to work in, and you are able to carry out the details of the operation on the septum, &c., with greater facility and accuracy.

MITRAL STENOSIS.

Clinical Lecture delivered at St. Thomas's Hospital.

BY

SEYMOUR J. SHARKEY, M.D.

STENOSIS of the mitral orifice is a condition which is often easily diagnosed at the bed-side; but nevertheless there are many cases in which its recognition is difficult, if not impossible, and one finds more frequently difference of opinion as to whether mitral constriction is or is not present, than one does as to the presence or absence of any other chronic valvular defect.

Anatomically the results of the gradual thickening and contraction of the mitral valve are revealed in the well-known conditions, the "button-hole" orifice and the "funnel-shaped" orifice. The former generally occurs when the flaps of the valve are pretty uniformly affected, so that the general shrinkage produces an oval or sometimes roundish orifice in the thickened curtain. In the case of the funnel-shaped orifice, the free edges of the valve are disproportionately thickened, and the flaps still jut out into the left ventricle, where the apex of the funnel is seen, while the insertion of the valve in the wall of the heart forms the larger extremity of the funnel. But such anatomical details are not to be made out clinically, and the best physicians are content with the diagnosis of mitral constriction.

When one examines a well-marked case of the disease, one notices on inspection that the apex-beat of the heart is in its normal situation, the meaning of this being that the left ventricle is not enlarged; indeed it may be diminished in size. This depends upon the fact that its work is lessened owing to the auricle not being able to discharge so much blood into it as it does in health. But this only applies to cases where there is no regurgitation, *i. e.* to cases of pure mitral constriction. In mitral regurgitation the auricle which is overfilled at great pressure by the blood poured back into it through the mitral orifice, discharges its contents again into the left ventricle with accumulated force, as soon as the ventricle ceases to contract; and the latter hypertrophies to meet the abnormal strain.

On palpation a thrill is felt at the end of the diastole, which is brought to an abrupt ending by a short, sharp cardiac impulse. The ventricle, receiving a small quantity of blood, can contract more rapidly than under normal conditions, and thus the short, sharp apex-beat is produced.

On percussion the cardiac dulness is found to be normal towards the left, and generally increased towards the right, reaching sometimes nearly as far as the right nipple. It may also extend upwards on the left of the sternum to the third or even to the second costal cartilage. These changes are due to an increase in the size of the left auricle, right auricle, and right ventricle, an increase which depends partly on dilatation, partly on hypertrophy.

Auscultation yields pretty definite and constant results in most valvular diseases. In aortic regurgitation, and in mitral regurgitation, for example, the murmurs heard in successive cases are not very different. But in mitral constriction it is far otherwise. The observer may hear—

1. A presystolic murmur; that is, a murmur which occupies the last third or half of the diastolic period only. Two conditions are essential to the production of the presystolic murmur. The first is a sufficiently narrowed orifice, so that in the earlier stages of constriction no murmur is produced. The second is a sufficiently high blood-pressure in the auricle, so that the stream of blood may be driven with force enough from the auricle into the ventricle to give rise to vibrations and produce the murmur. It may be that as the blood

passes through the mitral orifice the stream is not forcible enough to produce any sound until it is accelerated by the contraction of the auricle. It is under these circumstances that the presystolic murmur is heard.

2. But there is another period in the diastole in which the blood-pressure in the auricle may be great enough to produce a murmur, and that is at the commencement of diastole. The mitral orifice being narrowed, the left auricle gets distended with blood, and when the ventricular systole occurs it stops the inflow of the blood, and still further heightens the pressure in the auricle. As soon as the contraction of the ventricle is over, the blood flows onwards under very high pressure, and may give rise to an early diastolic murmur. As far as my experience goes, this murmur very rarely exists alone. When it is present it runs into the presystolic murmur, and consequently a murmur is heard continuously from beginning to end of the diastole. But in these cases the murmur, as a rule, becomes louder and more rumbling during the presystolic period. This point is of some importance in diagnosis, because (as I have proved post mortem) a continuous, even murmur, occupying the whole diastole, and produced at the mitral orifice, may be due to a dilated and not to a constricted opening.

3. In some cases, or at least at certain periods of some cases, a continuous murmur, uniform in character, may be heard during the whole of the diastole. The reason of this probably is that the narrowing of the orifice, and the force of the accumulated blood behind, are sufficient to give rise to such a murmur; but the auricular contraction is so enfeebled as to be unable to produce much acceleration of the stream in the presystolic period.

4. There may be no murmur. This may not only be the case in the earlier stages of constriction, but in the later also. The presystolic murmur, depending on auricular contraction, is loud when the latter is energetic, and may totally disappear when the auricle fails, and may reappear again as the heart's muscle recovers power.

Besides the murmur, some other points are of importance in auscultation. One is, that in a great number of cases of this disease the second sound is not heard at the apex. Another is, that the first sound is short and sharp, owing probably

to the ease and suddenness with which the left ventricle can contract upon its diminished contents. A third point is that the second sound over the pulmonary valves is accentuated. In doubtful cases of mitral stenosis this is of the utmost importance, especially where there is no other condition discoverable which could explain this phenomenon.

There is one other auscultatory sign which, in the absence of others, suggests to an experienced ear the presence of the disease. I described this years ago as "a peculiar hesitation in the production of the first sound, a kind of hanging fire, which is easier to appreciate with the stethoscope than to describe." This may be due to the prolongation of the diastole, for the blood goes through the mitral orifice slowly, and it takes a somewhat longer period of time to produce the degree of blood-pressure in the left ventricle which gives rise to its contraction.

The course of the disease.—This may be divided into two periods, the first that of good compensation, the second that of failing compensation.

During the first of these periods there is a moderate degree of narrowing of the orifice, and the auricle is not only dilated, but also hypertrophied. There may then be very few symptoms, and the disease may only be discovered, so to say, accidentally—that is to say, during the routine examination of a patient's organs who makes no complaints with reference to the heart; or there may be some shortness of breath on unusual exertion, or slight blueness of lips and cheeks. The pulse is generally small, and may be very infrequent. Indeed, well-marked bradycardia may be present in mitral stenosis, the number of beats per minute being as low as forty. In other cases the pulse-rate may be quickened.

In the second period, that namely of failing compensation, the auricular walls become enfeebled and the cavity of the auricle dilated, and the blood accumulates more and more behind the orifice, in the lungs, right heart, and general venous circulation. Then the pulse becomes small, feeble, rapid, and irregular, and many of the heart's beats, as many even as half, may produce no appreciable effect upon the radial artery. The frequency of the pulse can then only be safely estimated by auscultation of the heart. This fact is not always borne in mind, and the notes of such cases sometimes

give a very inaccurate record of the frequency of the heart's beat.

The blood being blocked in the lungs, respiration becomes more rapid, and dyspnoea or even orthopnoea occurs, accompanied by more or less cyanosis. Fluid may ooze from the distended pulmonary capillaries, causing oedema of the lungs, or hæmorrhagic infarction with hæmoptysis may occur. The latter occurrence is ordinarily the result of embolism of one of the peripheral pulmonary arteries by the dislodgment of a piece of thrombus formed in the cavities of the right side of the heart; but it probably also originates at times from primary thrombosis in the vessels of the lung.

The obstruction in the general circulation is shown by dropsy, occurring first in the feet, and then more universally. Before this occurs, however, the failure of the right side of the heart is often rendered evident to the careful observer by the enlarging and tender liver, and the examination of this organ is very important in chronic valvular disease, even from a prognostic point of view.

The diminished arterial pressure, and the stoppage of the free flow of blood through the kidneys, produces a diminution, often great, in the quantity of urine passed, which is concentrated and high coloured. It may also contain albumen and many casts. The latter are, however, hyaline, not granular or cellular, and only indicate obstruction in the renal circulation and not organic disease of the kidneys. The microscopic examination of many kidneys in persons who have died from cardiac dropsy has convinced me that mere venous congestion of these organs from heart disease does not give rise to serious organic disease, such as contracted kidney. Nor do I believe that the so-called "nutmeg liver" ends in cirrhosis. It is true that the connective tissue of these organs in chronic cases may look hard in outline and a little thickened, but serious cirrhosis of liver or kidneys does not result from venous obstruction. The latter, it must be remembered, may often occur in patients who already have cirrhosis from other causes. The spleen in mitral constriction is sometimes large and sometimes small, always hard, and may show areas of recent or old infarction, as also may the kidneys. When sudden infarction occurs in the latter organs, it is generally evidenced at the bed-side by hæmaturia with

or without lumbar pain, and cellular and granular casts may appear in the urine.

The altered circulation in the gastro-intestinal canal frequently causes troublesome dyspepsia, and sometimes hæmorrhage from the bowels; while the obstruction in the cerebral circulation may produce serious insomnia, and sometimes delirium. Occasionally a fibrinous embolus from the left auricle blocks one of the cerebral arteries, and produces limited or wide-spread softening.

In such cases of general dropsy it now and then occurs that some large vein, say the axillary or femoral of one side, gets blocked by a local thrombus, and then the swelling, pain, and tension in the corresponding limb are much greater than elsewhere. A patient, too, who can only lie on one side may thus get an unsymmetrical dropsy owing to gravitation.

Certain physical signs may occur in connection with the heart when the stage of failure sets in, which are often of importance in estimating the degree of failure which has occurred, or in foreshadowing its occurrence. Thus, the presystolic murmur may disappear owing to the complete or partial failure of the contraction of the left auricle, and a sign of similar import is the diminishing intensity of the second sound on the pulmonary side. The more complete the stasis in the pulmonary circulation and left auricle the less the quantity of blood which flows through the pulmonary orifice at each contraction of the right ventricle. The semilunar valves therefore undergo less movement, less sudden change from flaccidity to tension, and the sound they produce diminishes in intensity. In the stage of failing compensation the left ventricle may gradually hypertrophy, as the tension in the arteries which it has to overcome increases owing to backward pressure.

The appearance of a tricuspid murmur, and pulsation in the jugular veins, or even in the liver, may testify to a dilated tricuspid orifice allowing of regurgitation.

The patient, whose distress gradually increases from so many causes, may gradually die in spite of all the efforts made to save him. On the other hand, the treatment of such cases is as a rule very hopeful, and although the actual narrowing of the mitral orifice must still remain, and may even increase, death may be averted and years of fairly comfort-

able and useful, though quiet, life be secured. Under favorable circumstances, the dropsy gradually disappears, the urine increases in quantity, and albumen and casts disappear from it, the liver gets smaller and less tender. Respiration becomes easier, the pulse slower, more regular, and fuller, the presystolic murmur reappears, and the second pulmonary sound becomes louder. The patient sleeps better, eats and digests his food better, and gradually returns to a condition of comfort.

Treatment.—The first and most essential point in the treatment of heart failure in cases of valve disease is rest; the patient should be put to bed. This measure of itself sometimes works wonders. If the venous congestion is very great, bleeding should be resorted to in cases of mitral stenosis.

As regards drugs, digitalis still holds the foremost place where the pulse is rapid, feeble, and irregular, and in most cases its effects are very striking. But it is useless to give it with a slow pulse, that is to say, where the pulse is beating at a rate which is normal to the individual in question, or below that rate. We cannot hope to increase the force of the circulation as a whole if, while we add to the power of each contraction of the heart, we considerably diminish the number of beats per minute; unless, indeed, when we begin to give digitalis the heart's beats are abnormally hurried as well as feeble. Occasionally under such circumstances digitalis fails, and strophanthus may then be tried; but the latter drug is far less frequently effectual than digitalis.

Combined with digitalis we may administer strychnine, which is one of the best cardiac tonics we possess, especially when given subcutaneously. It may be given when the heart's action is slow, and it may be continued after digitalis has reduced the frequency of the pulse. Another very good heart tonic, especially when the urine is diminished, is citrate of caffeine.

Alcohol should also be given in moderate quantities.

The bowels should be kept well open, but purging is not advisable.

Dyspepsia generally depends on the congestion of liver, and gastro-intestinal mucous membrane, and is relieved by the measures which improve the heart's action; but bicarbonate of soda with a bitter infusion often aids in the re-establish-

ment of the gastric secretion. Abundant simple food in small quantities, often repeated, is an essential in successful treatment.

One of the most difficult complications to manage successfully is insomnia. Morphia by the mouth or in small quantities subcutaneously is often successful. Small doses of chloral may be given with caution, and should be administered with some alcohol or other cardiac stimulant. Paraldehyde, chloralamide, sulphonal, and other narcotics may also be tried, but they frequently fail.

After the more urgent symptoms of cardiac failure have subsided, Schott's treatment may produce very good results; but the time at my disposal prohibits any further consideration of this method of procedure, and of many other important points in the treatment of these cases.

A DEMONSTRATION ON A CASE OF APPENDICITIS.

Delivered in the Wards of St. Bartholomew's Hospital

By C. B. LOCKWOOD, F.R.C.S.,

Assistant Surgeon to the Hospital, Surgeon to Great Northern Hospital.

GENTLEMEN,—A few years ago cases of this kind were seldom seen in the surgical wards. You must not, however, think that because they are now sent to the surgeon that they all call for operation. Many cases of appendicitis recover after the first attack, and afterwards remain well. Others, as in the present instance, require to be carefully watched by the surgeon in order that the proper moment for intervention may be seized. This patient, you will observe, is a strong and healthy countryman, who is 26 years old; his expression is anxious, and he appears to be in pain. The statement that he has had an attack of appendicitis is borne out by the history of his complaint. Ten weeks ago he was taken with a sudden pain in the right side of the abdomen, at the same time he says that he was constipated, and passed for a time no fæces and very little

flatus, and his abdomen became slightly distended, he felt sick but he did not vomit; these are the symptoms of slight intestinal obstruction. When the vermiform appendix is inflamed the intestine in its vicinity is likewise affected, and ceases to perform its functions properly. One of the most important of these functions is the propulsion of its contents.

This inflammation of the intestine in the neighbourhood of the appendix has, in my opinion, a very important relation to the treatment. If purges be given by the mouth to overcome the constipation they often cause considerable suffering by the disturbance which they excite in the inflamed bowel. Sometimes they may, indeed, fail to pass through it, in which case they are a source of great suffering and considerable danger. Therefore, when an operation is unnecessary it is best to overcome the constipation by enemas, from which I never remember to have seen any harm ensue. Besides the treatment by enemas, it is but humane to administer moderate doses of opium for the relief of pain.

If now you look at the patient's abdomen you will notice that it is still a little distended, and when he breathes the right side hardly moves. Gentle pressure with the finger is borne without flinching in every part except where it is held still. We will press upon the situation of the vermiform appendix where it lies beneath the right linea semilunaris in a line drawn from the anterior superior spine of the ilium to the umbilicus. At this point there is a distinct induration, and perhaps we can feel a hard swelling. Part of this induration is caused by the contraction of the abdominal muscles; these always become hard and rigid over inflamed organs to protect them from any kind of violence or even from pressure. The lump which can be felt is usually said to be the inflamed appendix, but this is, I believe, seldom the case; when removing the inflamed appendix it is usual to find a mass of thickened and indurated omentum, together with inflamed and thickened intestine, with the appendix hidden away behind them, so that it could hardly have been felt. Now observe particularly the position in which this patient is lying; he reclines upon his back with his right hip held flexed upon the abdomen, and any attempt at extension is resisted because it causes pain. This is probably accounted

for by the position of the inflamed appendix. Lying beneath the cæcum in the iliac fossa, it causes an inflammation of the peritoneal lining of the iliac fossa, of the iliac fascia, and of the iliacus muscle. Before we assume that this inference is correct we will make a rectal examination. Not infrequently the end of the inflamed appendix hangs over the external iliac vessels into the true pelvis. Under these circumstances the inflammation in it often spreads to the peritoneum, covering the bladder, and probably to the wall of the bladder itself, causing increased frequency of micturition, or, as in a case I recently operated upon, complete inability to empty the bladder. It is, therefore, of great importance to inquire into this point. You will note that this patient has had no trouble whatever with his urinary functions. This helps to confirm our inference that his inflamed appendix lies in the iliac fossa, and does not hang into the true pelvis. But, in addition, the finger in the rectum cannot detect it in that position. The occasional presence of the appendix in the pelvis may also lead to the formation of an abscess in Douglas's pouch, but of this there is no evidence in this case. I have seen many collections of pus in Douglas's pouch entirely overlooked because no rectal examination was made; I cannot sufficiently impress upon you the extreme importance of this step, or the discredit which its omission entails. It is hardly possible to say what is the exact condition of this man's appendix. During the ten days that he has been in this hospital his temperature has not risen above 100° F., but it would be rash on this account to say that there is no suppuration around the appendix. On the contrary, if you count his pulse you will find that it is slightly accelerated, and is beating nearly ninety times per minute. This is rather rapid for a stolid countryman, and makes me suspect that there is probably an abscess.

Inasmuch as his illness has now continued twelve weeks, and as we have observed that the pain and tenderness and swelling had slightly increased, I propose now to accede to his wishes and perform an operation for the removal of his diseased appendix. We must not, however, be always too positive about the diagnosis of such cases as these. Two years ago I operated upon a youth who had had what everyone considered to be two typical attacks of appendicitis. When the

abdomen was opened it was found that he had acute tuberculous peritonitis which was localised in the cæcum, in the vermiform appendix, the end of the ileum, and the mesentery; no harm whatever followed this operation, but rather good, because it entirely cured the pain which had previously been severe. Afterwards he died of acute general tuberculosis. On another occasion I operated upon a lady who had had what highly competent observers considered to be two attacks of acute appendicitis. I cut down upon a lump which could be distinctly felt in the position of the appendix, and with great difficulty, found that organ encircling the end of the ileum and adherent to it and to the front of the mesentery. When it had been removed there still remained a lump the size of a walnut at its origin from the cæcum. This lump had a most suspicious appearance, and a piece of it was removed for examination and the rest covered over with peritoneum. A histological examination was made, and proved that it was a carcinoma. Shortly afterwards I resected this mass together with the end of the ileum and cæcum, and the patient made an uneventful recovery. I ought to add that Dr. Craig, who was in charge of this case, suspected its nature, being mainly guided by the family history.

The operation upon the patient we have just examined will be performed in the following manner:—An incision three inches long will be made one inch above and parallel to the outer half of Poupart's ligament; as a rule this will suffice, but if necessary it can be prolonged at either end. To do this it may be necessary to divide the deep epigastric vessels, and although I have often had to do this I have seen no ill result follow. When the abdominal walls are cut through great care is necessary in opening the peritoneum; it is often inflamed and thickened, and hard to recognise, and it may be adherent to the cæcum or intestine. After the peritoneum has been opened the cæcum is pushed aside and the appendix sought for. This may prove to be one of the most difficult tasks that a surgeon can undertake. As a rule the appendix is hidden under the end of the great omentum, which is adherent to it. Sometimes it is bound down by a mass of inflammatory adhesions.

Not long since I operated upon a young gentleman who had had four attacks of catarrhal appen-

ditis; the most careful search revealed no appendix. The sense of touch afforded but little information where so many structures had become indurated by inflammation, and where the enlarged lymphatic glands could easily have been mistaken for it. The anterior longitudinal muscular band gave no assistance, although it was carefully traced downwards to see if it led to the root of the appendix; and perhaps I may add that I have received less assistance from this device than might have been expected, because, owing to the inflammation, it is impossible to trace the anterior longitudinal band as can be done in the dead-house or dissecting room; moreover, no trace of the appendix could be found at the ileo-cæcal angle, where its root is generally to be found. But some years ago I showed specimens at the Pathological Society in which the vermiform appendix was hidden away in one or other of the fossæ about the cæcum, and also, in conjunction with my friend Dr. Rolleston, published a paper in the 'Journal of Anatomy and Physiology' in which every variety of this abnormality was described, and therefore I proceeded to ascertain whether in this case the appendix might not be concealed in one of these retro-peritoneal pouches. I saw a delicate streak running across the peritoneum of the iliac fossa; when this was searched with a pair of dissecting forceps the peritoneum came apart and revealed the distended appendix lying hidden away in the ileo-cæcal fossa. I do not believe that the vermiform appendix is ever absent except as the result of disease. In the numerous cases in which operators have failed to find it, it had many times escaped their search because it was hidden in a fossa, as in the case I have just described. Twice at the Great Northern Hospital this has occurred, the appendix being found after death hidden in a retro-peritoneal fossa. Moreover, the appendix itself may be difficult to recognise. Highly competent surgeons have told me that in performing this operation they have been in constant dread of mistaking the external iliac artery or the ureter for the appendix. When once the appendix has been found and separated, the rest of the operation is easy; its mesentery is clamped, secured with a ligature which transfixes it, as all pedicles should be transfixed, and divided. The peritoneal coat of the appendix is cut through and turned back for half an inch; the muscular and

mucous coats are tied in two places with a fine silk ligature, and divided between. If no ligature or clamp be applied, some of the contents may escape and infect the wound. Next, the interior of the stump of the appendix is touched with pure carbolic acid, or with a cautery, and the peritoneum, which was cut long for this purpose, is brought over it with a couple of points of Lembert's suture. The iliac fossa having been cleansed of blood, the wound is closed with a row of fishing-gut sutures. If the abdominal wall is very thick and muscular, it may be brought together layer by layer with buried sutures. If no pus or septic fluid is met with in the course of the operation it may not be necessary to drain, but, as in all other operations, I always drain if anything septic has entered the wound.

Cases of appendicitis present, of course, great variety. This depends in a great measure upon the kind of disease which is in progress in the organ. During the past two or three years you may have seen me operate on cases in which the peritonitis was violent and acute, and had rapidly become general; in others in which the peritonitis was very acute, but had led to the formation of a localised collection of pus. On others, again, like in this, where the peritonitis was localised but more chronic in its course, and lastly, in cases of recurrent appendicitis in which there was an interval of apparent restoration to health. When these latter are operated upon they do exceedingly well, and give hardly more anxiety than an ordinary case of radical cure of hernia; indeed, it is usual for the patients themselves to say that the operation does not cause them so much pain and suffering as one of the attacks. The chronic cases, such as the one we have now before us, also do exceedingly well, and are hardly more dangerous as regards operation than those of recurrent appendicitis. But those which are acute with localised collections of pus are more anxious and dangerous, and in my experience the safety of the patient depends upon the promptness of surgical intervention. Twice within the last four months I have opened acute abscesses of this kind, and removed from them faecal concretions which had escaped from an ulcerated or gangrenous appendix. Surely it cannot be right to delay in cases of this kind. I need hardly say that when the peritonitis has become diffuse the position of affairs is well-nigh

desperate, but not absolutely hopeless.* Therefore we anticipate that after the operation upon this man he will not have a serious illness or give rise to much anxiety. As in other abdominal cases, he may first of all have troublesome vomiting. To avoid this, we shall, during the first twenty-four hours, be exceedingly careful with what is given him by the mouth. As a rule, occasional sips of hot water are given to relieve the thirst which is always so distressing. Should it be necessary, we may give him enemata of tepid water, with the same object in view. If he suffers from shock to any dangerous extent, enemata of brandy will be given, likewise hypodermic injections of strychnine ($\frac{1}{50}$ grain). Feeding by the mouth will be begun with great caution. I am accustomed to begin with a tablespoonful of meat essence the first hour, a little brandy and soda water or champagne the second hour, and a table-spoonful of peptonized milk flavoured with a little tea or coffee or brandy the third, and then continue in the same way. If there is no vomiting, the quantities are increased and given at longer intervals. In cases of vomiting our reliance is placed on rectal feeding, which is in itself an art. I have not time to go into this most important matter. It is a subject which you ought to thoroughly master. But I would say that these enemata should be highly nutritious, concentrated, peptonized, and unirritating.

A useful formula for nutrient enemata is equal parts of good milk and meat or mutton essence peptonized for at least twenty minutes with Benger's liquor pancreaticus, with the addition of a table-spoonful of really good brandy to each ounce. As a rule three to four ounces of this enema can be retained and absorbed every three hours. It should be administered with a No. 9 soft rubber catheter. Four or five minims of tincture of opium may have to be added to help the rectum to retain it. If the anus is sore a little solution of cocaine adds materially to the comfort of the patient, but everything depends upon the skill with which it is given. Some of our sisters and nurses are so efficient that they can continue rectal feeding for many days, or even weeks. After vomiting the patient's trouble is usually flatulent distension.

* See 'The Surgical Treatment of Diffuse Septic Peritonitis; with successful cases,' by C. B. Lockwood, 'Med.-Chir. Trans.,' vol. lxxviii, 1895.

Of course the operation excites a certain amount of inflammation around the end of the ileum and cæcum, where the intestinal canal is narrow, and where, as I have already pointed out, inflammation easily impedes its function of propelling its contents. In addition the abdominal wall is painful after having been incised and sutured, and the patient cannot use it to help to expel the intestinal contents. Under these circumstances I am accustomed to order an enema for an adult of half an ounce of castor oil and half an ounce of turpentine mixed in eight or ten ounces of thin gruel. This usually succeeds after one or more attempts. Its action is often aided by the insertion of the rectal tube. At the same time there can be little doubt but that a hypodermic injection of strychnine also helps to overcome and prevent this flatulent distension. As soon as the dangers of vomiting and flatulence are passed, it is better, as soon as possible, to begin a solid dietary. I have observed that patients do better and are more comfortable when they are spared the infliction of quantities of fluid, especially milk. After the operation it is desirable that this patient should lie in bed for three weeks, and afterwards remain recumbent on a couch for another three weeks, in order that his abdominal wound may become strong and secure. After that it is undesirable that he should do any hard work for another six weeks. In other words, he must do exactly as our cases of radical cure of hernia do.

P.S.—The operation which has been described above was performed three days after this demonstration. The peritoneum was found to be thickened when the abdominal wall was incised. About an ounce of pus escaped from a small abscess cavity near the appendix. Some adherent omentum was tied and divided. The vermiform appendix lay in the iliac fossa, coiled upon itself and adherent to the small intestines. After it had been removed, in the manner which has been detailed, its end was found to be perforated by an ulcer. No fæcal concretion was seen. The abscess cavity was disinfected with 1 in 1000 biniodide of mercury lotion, and a drainage-tube inserted and the wound closed with fishing-gut sutures. The operation was followed by no shock. The temperature never rose above normal. His bowels were opened by an enema on the third day. Some pus continued to escape from the wound for two days. The drainage-tube was

removed on the fourth day, and at the end of a fortnight half the stitches were removed and the rest at the end of the three weeks, when the wound was healed.

NOTES.

Iodvasagen as a Substitute for Internal Administration of Salts of Iodine.—Leistikow ('Monatshefte für Praktische Dermatologie') calls attention to the fact that the mouth administration of the iodides of potassium, sodium or rubidium is attended with marked disadvantages, since it is liable to occasion catarrhal involvement of the mucous membrane of the mouth, nose, stomach, pharynx, and bronchial tubes, or rheumatoid pains, palpitation of the heart, and iodine dermatitis. Under such circumstances it has been the custom to abandon iodides, using mercury instead, or to employ enemata of potassium iodide. This latter method of administration is not one to be recommended, since, even if the rectum tolerates the drug, the systemic effects are the same as those produced by mouth administration. He has, however, had excellent results in the administration of the drug in the form of iodvasagen inunctions. In all he has employed it in twenty of his private cases. There are two preparations, one containing six, the other ten per cent. of iodine. The former is to be preferred, since it does not produce follicular inflammation. The application is made exactly as is the mercuric inunction—forty-five grains of iodvasagen are daily rubbed in for ten minutes; the first day the left arm being selected; the second the right arm; the third the breast; the fourth the belly; the fifth the left leg; the sixth the right leg. The rubbings are continued for about three weeks. In four cases the rubbings had to be discontinued because of violent naso-pharyngeal catarrh. The remaining cases showed no reaction whatever, and the specific symptoms rapidly disappeared. From his experience Leistikow draws the conclusion that iodvasagen inunctions are an admirable substitute for the internal administration of the salts of iodine.—*Therapeutic Gazette*.

Cancer of the Prostate.—The curative surgical treatment of cancer of the prostate has not yet been determined, the two principal reasons for which are: The difficulty of a correct diagnosis, at first, and the uselessness of active intervention when it is too late, when diffusions are already present. Only a purely symptomatic treatment can be of use. Without being able to conjecture the importance of an early diagnosis, from a point of view of curative intervention, there would still be an advantage in being able to make a diagnosis in the early stages of the malady; it is from this point of view that Guepin's work derives its importance, throwing, as it does, new light upon the subject. Tumours of the prostate usually take their point of inception in the glands, presumably remaining localized at that place for some time, giving rise to phenomena of stagnation and retention of the secretions in the glandular cavities, or to other symptoms, resembling those presented by senile, hypertrophied prostate; these symptoms later are noticeable in the ganglia and the neighbouring regions. It is a question of prime importance to be able to recognise these inceptive prostatic neoplasms in old men with enlarged prostate glands. The method to pursue, as first pointed out by Guepin, after obtaining the objective and the subjective signs of hypertrophied prostate, is given in the author's own words: "To my mind, a presumed sign of the existence of a cancer in old men affected with stagnation of urine and elevation of the vesical neck is the presence, in one of the prostatic lobes of a nodule, giving, by digital sensation, the impression of a hard cyst, remaining so after the treatment and cure of the stagnant secretions and the congested organ."—*Medical Fortnightly*.

"SAXIN" (B. W. & Co.) is a powerful sweetening agent issued in "Tabloid" form; and is about six hundred times sweeter than sugar. A $\frac{1}{4}$ gr. "Saxin" "Tabloid" may be substituted for each lump of sugar in the case of patients suffering from diabetes, gout, obesity, glycosuria, &c.

A NEW work on heart disease entitled, "Cardiac Failure and its Treatment, with especial reference to the use of Baths and Exercises," by Dr. Alexander Morison, is in the press, and will be issued shortly by the Rebman Publishing Company.

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SEBORRHŒA AND SEBORRHŒIC DERMATITIS.*

BY

RADCLIFFE CROCKER, M.D.

THE subject of seborrhœa has always been one of importance, consciously or unconsciously, to the great army of men who become more or less bald. But of late years it has acquired an additional practical importance to all medical men who have anything to do with common diseases of the skin, since it has been shown that it is an important ætiological factor in the production of many forms of inflammation of the skin, and the researches of Duhring and Unna, and many subsequent workers have shown that these are no longer to be lumped together with eczema, with psoriasis, and with various forms of lichen. I should like to first discuss what is really meant by seborrhœa, and what some very recent researches have shown its nature to be. Naturally the first idea was that seborrhœa connotes an increase of the sebaceous secretion, but as used in modern dermatology it connotes something more than that; there is not only an increase of secretion, but an alteration in the nature of that secretion by the addition of numerous epithelium cells, and these, with the other changes, lead to the atrophy of the hair and papillæ, as I propose to explain presently. I take first what we call a marked condition of seborrhœa. We find the scalp is covered with a fatty crust, yellowish and almost waxy in consistence, and adhering rather firmly to the scalp, but which may be detached by any blunt instrument or the finger-nail, and the skin beneath is found to be apparently perfectly healthy. But it is noted by the owners of this condition that the hair begins to come out with the ordinary manipulations of combing and brushing, and otherwise cleansing

the hair, beginning chiefly in the crown of the head and the temporal region; the temporal line recedes, the hair on the top becomes gradually thinner. First of all the displaced hair is replaced by poorer and thinner hair, then the hair ceases to be produced, the bald area increases in size, and the parting which none of us like to part with, gets more and more lateral until at last, like the pen of a clerk, it is suspended above the top of the ear. This we are all familiar with. This is a very marked and typical condition of seborrhœa; but frequently the proportion of fat in association with this condition varies considerably, and so the scaly element is sometimes more prominent, the scales less and less fatty, and sometimes there is an opposite extreme, the white powdery scales falling upon the clothing to the extreme annoyance of the patient. It is a condition which is common in elderly men, and not infrequent in young ones.

I show you a very old plate illustrating a condition of seborrhœa in what I may call an ordinary case. The scales are dirty-looking but not so black as the plate suggests. In this stage and condition, all external signs of inflammation are wanting, though microscopically they are to some extent present. It has long been inferred on clinical grounds, and our increasing knowledge of what microbes will do support this view, that it is a microbic affection, but, owing to the numerous microbes which are always found in the head, the exact microbe has not been isolated. But I am fortunate enough in being able to put before you some of the most recent researches into seborrhœa, made by a young Frenchman named Sabouraud, who has done some extremely good work on the fungi of ringworm, and who has latterly turned his attention to alopecia areata, and to the baldness produced by seborrhœa. These researches have only been quite recently published. He explains scientifically what we had already inferred clinically, and we now have it before us in a definite manner. I show you a diagram illustrating the changes which take place in the hair in seborrhœa. The seborrhœic microbe gets in at the orifice of the hair follicle, and travels

* An Address delivered before the Thames Valley Branch of the British Medical Association.

downwards till it reaches the sebaceous gland. There these microbes increase in number, and by their irritative presence produce epithelial cell proliferation, which forms around them a pseudocyst, which does not surround the hair, but usually forms a projection upon the side of it. In addition to this pseudo-cyst it excites proliferation of the lymphocytes, which are depicted here, together with some giant cells in one group surrounding the papilla, but not entering into it, some going into the angle of the sebaceous gland and the arrector muscles, and others just round what he calls the cocoon of the seborrhœic microbe. Similar cells surround the vessels in the immediate neighbourhood, but there is nothing as yet on the surface to indicate it. As a result of this cell infiltration, the nutrition of the papilla is very much interfered with, the first effect of which is that the pigment of the cells immediately surrounding it becomes extravasated, the old hair is thrown off, and the new hair which replaces it is formed without a medulla, and to a great extent without its pigment. In fact we get an adult hair replaced by an infantile one. If the process goes on, the resulting damage is still greater to nutrition, and ultimately no hair at all is formed. Now, Sabouraud says that this mechanism is not absolutely peculiar to seborrhœa, but that we see exactly the same condition anatomically, and he believes the cause of alopecia areata is this same microbe, but that is another story. He also says that the comedo which we are so familiar with is really made up of an assemblage of these seborrhœic cocoons as he calls them, together with numerous abortive hairs, all enclosed in an epithelial covering. That part also I will not go into now, but will go on to the chief subject of my discourse. People who are subject to seborrhœa are liable to get forms of inflammation upon the skin which, for a long time, were much misunderstood. These (showing plate) are all examples of a papular eruption which is found most typically in the interscapular region, and also in the centre of the chest. Here again is an illustration of an extreme case where the whole front of the body is affected, but in the great bulk of the cases it is limited to the regions I have mentioned. It very rarely comes down upon the limbs. You will practically always see the eruption composed of little groups of minute pin's point papules, forming circular patches which tend to enlarge peripherally, clear in the centre, and

leaving a stain such as is shown here. Thus it is very much like *tinea versicolor*, but always with an inflammatory papular edge. That condition, which has been called *lichen circumscriptus* and various other names, was formerly shown by Duhring to be constantly connected with this scurfy condition of the scalp. The practical point for you to remember is that whenever you see an example of this eruption you should examine the scalp, for although the eruption is easily removed for a time, it is extremely liable to recur, and patients constantly come saying that they have something which takes it away for a little time, but as soon as they leave off the remedy it comes back again. The theory is, that the patient in brushing his hair, brushes some of the microbes from the scalp, and plants them into the sebaceous orifices in the back, and on the chest, and the rational treatment is therefore to treat the condition of the scalp, as a means of bringing permanent cure for this eruption, which, although not very important in itself, is one which occasions a good deal of annoyance and anxiety to the patient. This we now speak of as *seborrhœa papulosa*, instead of *lichen circinatus* or *circumscriptus*, and it is one of the forms of seborrhœic dermatitis, and one of the most common forms.

Its treatment is by a mild antiseptic ointment—thymol twenty grains to the ounce of lard; or sulphur ten grains to an ounce of boracic acid ointment. These and many others which will occur to you should be rubbed in, and will soon remove the eruption on the skin, and you must then also treat the scalp effectively to get a permanent result.

In some cases, instead of imitating a form of lichen it may simulate the eruption of psoriasis, and you get patches of well-defined inflammation standing strongly out against the healthy skin, and more or less covered with scales. I have brought as a means of comparison some plates of the ordinary forms of psoriasis. You will see that although there is a resemblance there are several very considerable differences. First as regards the locality, the psoriasiform seborrhœic dermatitis occurs mainly in the axillæ, the sides of the body, the groins, and the trunk generally; then there are patches upon the scalp. Although the eruption resembles psoriasis very closely and the inflammation is a dry one, there is not the crust of silvery scales such as we see in typical psoriasis; the

scales in seborrhœa are comparatively scanty and more or less fatty. You see it does not particularly attack the extensor aspects of the limbs; in fact, the limbs, as a rule, very often escape entirely. Besides the general distribution—which is different, in the patches themselves, though the similarity is considerable—there are very marked differences. First, in the absence of the heaping up of silvery scales, their scantiness and fatty character, and secondly, when the scales are removed with the nails or otherwise, you do not see these bright red vascular bleeding points which are so characteristic of ordinary forms of psoriasis. The psoriasiform eruption is not quite so common as either the papular form or the eczematous-looking eruptions of which I now show you some examples. This plate shows another papular form of seborrhœa. It differs, perhaps, from the first form in that there is not the same tendency to group, nor the same tendency to localise in the interscapular and presternal region; it forms around the follicles, very often is distributed widely over the trunk, and may run into scaly patches. It represents what was formerly called lichen simplex, which we now know to be really a seborrhœic papular dermatitis.

Now let us revert to seborrhœa. I showed you just now that very frequently in seborrhœa there is a complete absence of any sign of inflammation in the ordinary form such as leads to baldness, but under certain adverse conditions inflammation is excited to a greater or less degree, and sometimes to a very intense degree. This illustration shows a patient who had suffered from seborrhœa in its ordinary form for many years, the scalp was covered with dense scaliness, and the hair became very thin on the top, then she had considerable mental worry and trouble, and depreciation of her general health, and an acute inflammation started on the top of this, and produced the condition which you see represented. Here you see the inflammation extends beyond the scalp, but only to a moderate extent; there is a very sharply defined line of redness of the surface covered with fatty scales, mixed more or less with inflammatory exudation. This represents a condition which is really an acute seborrhœic eczema. There is no doubt a true eczema grafted on to the seborrhœic inflammation, when all the signs of eczema are modified somewhat by the seborrhœic condition,

especially with regard to the sharply defined line. In the majority of cases of seborrhœic eczema the line of demarcation between healthy and diseased skin is sharply defined, whereas in ordinary eczema it shades off almost imperceptibly. Such a condition as that would therefore require general treatment as well as local treatment, but all these other forms, as a rule, require no internal treatment, because they are due to local implantation of a microbe, and, therefore, they require only antiseptic applications to destroy the vitality of that microbe. If, however, there is any acute inflammation you must treat it as you would any other acute inflammation. *Do not mind what the name of the skin disease is*; if you have an acute inflammation, treat it with soothing and protecting and *continuous* applications. It is very often said that the bugbear of diseases of the skin is the multiplicity of names, but, gentlemen, the point really is not that. In a great number of instances you need not bother yourselves very much about names, provided you recognise the actual conditions that are present, and one case of a common psoriasis may stand tar or various other stimulating applications, and another acutely inflammatory psoriasis may have to be treated just as acute eczema would have to be treated. Therefore, you will understand that although these forms of seborrhœa have a microbic origin, you must also bear in mind the inflammatory condition in adopting your treatment to it. Now, the best way is to combine soothing and protecting applications with various antiseptics. Such a condition as I show you here would probably require an application of boric acid combined with loretin, iodoform, or other pus destroyers; eucrophen, loretin, and aristol are all iodoform substitutes, not, perhaps, as absolutely as good as iodoform, but owing to the compromising character of iodoform preferable from the patient's point of view, and undoubtedly preferable to the surrounding friends. The best of these substitutes, in my opinion, is loretin, and for such a condition as I show you here I should probably order twenty to thirty grains of boric acid, five grains of loretin, with perhaps the addition of some little lanolin. Or again, you can combine with loretin, oleate of zinc and an astringent application, as well as the antiseptic, but in any case the application should be arranged so that it will be continuously applied.

If the inflammation is very acute, glycerole of the subacetate of lead is useful. Various other things of that kind will occur to you, if you really understand what it is you are aiming at. I show you an illustration of what is called eczema rimosum. In this condition almost every time the hand is opened or shut the palm cracks. Unna claims that all these cases are of seborrhœic origin, and I think it must be admitted that there can be no doubt that a certain proportion of them are so. In the case of the woman whose picture I described to you just now, eczema rimosum was present on her palm, and this condition of the palm was the most troublesome to get rid of. Here is another form of seborrhœic dermatitis which is sometimes called eczema, somewhat allied to this condition but very much less acute. Here you see what is the characteristic of many of the seborrhœic inflammations, namely, a tendency to form rings and circles ever widening with well-defined borders. You see here dense scaliness of the scalp, and very little inflammation beyond it. She had a moderate degree of inflammation, with circles tending to spread almost like ringworm, but attended with very much more cell change and proliferation than is associated with the ordinary form of tinea tonsurans or circinata. The tendency of these eruptions is to attack the scalp and extend more or less beyond the borders of the hair, and, if I remember rightly, this patient had the whole of the eruption confined to these regions. Sometimes, however, they also have ringed eruptions on the trunk as well. Here you may use more stimulating applications than for a case of the first kind I showed you, always applying some microbe destroyer, and, as for example, a weak sulphur ointment, ten grains to the ounce, associated with zinc or lead ointment. She probably would not require any internal treatment. But in a patient broken down in mind and body, a good deal of internal treatment would be required as well. In some cases the inflammation on the skin is very slight, and in the case I show I have no doubt that the seborrhœa began in the face itself. The face is almost as frequently attacked with seborrhœa as the scalp itself, and in one form of it we would get what we may call pure, unadulterated seborrhœa, seborrhœia olesa—an increase in the oleaginous secretion without any other cell changes, but in her cases there are more or less cell changes and

a moderate amount of inflammation, situated symmetrically upon the cheeks, very much in the same position as you see in acné rosacea. That distribution is common to many forms of inflammation, and the case I show you here yields to very mild treatment, and generally boric acid ointment alone would get it well. Tar is sometimes useful in low-grade inflammations, but of course you must be careful in using tar on the face, only beginning with three or four drops of liquor carbonis detergens to the ounce, and feel your way as to using it stronger. Another form of inflammation which we see on the body is that which I show you here. This form attacks not only the trunk, but the limbs and other parts. You see that there is a tendency to preserve well-defined circles, with the border more raised and defined than the centre, and a tendency to enlarge peripherally, while the centre undergoes partial, or seldom complete, revolution. In other cases the inflammation may remain fairly uniform over the whole patch. A thing which should attract your attention is the well-defined borders which, if there were only one patch, would lead you to think it was a case of ringworm. But the duration of perhaps only a few weeks and the enormous extent would show you that it could not be merely a tinea, as we know that spreads much more slowly than these micrococcic forms of inflammation. However, when you see well-defined borders with what in other respects appears to be an eczema, think whether it is or may not be of microbic origin. One point is to investigate the scalp, and see if the condition of the scalp may not have some ætiological bearing on the condition of the body. When the eruption occurs away from exposed situations we may have to resort very frequently to lotions of an antiseptic character, which clean the skin like thiol and ichthyol, or to resorcin; for instance, ten to fifteen grains thiol, five grains resorcin, and a little compound tragacanth powder to one ounce water, painted on is often extremely advantageous. Or you may use picric acid or tar lotions, more or less diluted, according to the amount of inflammation you have to combat. Here, again, no internal treatment is required; you need not cut off all that the patient likes best, for gout has nothing whatever to do with it, and as long as he does not go in for excess of alcohol he does not require that strict anti-gout treatment which every inflammation

is almost sure to get nowadays. It is quite true, however, that patients are so educated up to believe that dermatitis means gout that they expect you to cut them off something. If you see that is the case, it is a pity to disappoint them ; but understand clearly that it is not necessary for the treatment of the disease itself.

To go back to treatment of seborrhœa of the scalp ; here the applications are again on the anti-septic tack, but you will find that the seborrhœic microbe, like *Tricophyton tonsurans* and others of the class, dislike acid media, therefore, as our object is to make ourselves as unpleasant to the microbe as possible, we combine acetic acid with other applications for the condition, and they are found very successful ; in addition to this acid you can give antiseptics, of which, perhaps, the most useful are resorcin and naphthol.

An example of such would be ;—Acetic acid half an ounce to one ounce, resorcin a drachm, eau de Cologne two ounces, rose water to eight ounces, glycerine 3j if the hair is very dry. But in many cases patients object to anything in the way of glycerine because the fashion now is to have fluffy hair, and glycerine prevents anything of that kind. But this by no means completes the story ; many other remedies will occur to you and may be substituted in different cases. But the application should be thoroughly applied. You know that half-antiseptics are no antiseptics ; many of these lotions are known to hairdressers, but they neglect to thoroughly apply them. Inculcate that the application should be rubbed in especially in the crown of the head and the temple, for there seborrhœa flourishes, probably because the scalp is thinner here, the vascularity less, and the nutrition probably somewhat diminished, and therefore the resistance diminished also. At all events, the fact remains that the microbe grows there with the greatest energy. One application per day will be sufficient, but it should be continued for at least six weeks to two months without intermission. The common cause of failure is that the patient starts off energetically for the first week or two, and then one night he says he is tired and will not do it that night and so on, and the result is practically complete failure. If it is continued uninterruptedly for six weeks or two months you may completely destroy the vitality of such microbes as remain. There is

no doubt that some people are much more vulnerable to this microbe than others, and it is practically certain that unless treatment is continued it is sure to be affected again. It is hard to say when we have destroyed the last microbe in any disease. Mercurial applications are also useful. Very often mercurial pomades are given after the patient has washed his head.

These are the principles upon which you should go in the treatment of seborrhœa. It is apparently a trifling disease, but it is important for you to realise that it often excites inflammations of the skin which are often of an important character, both as regards diagnosis and treatment.

DEMONSTRATION OF CASES AT CHARING CROSS HOSPITAL,

By STANLEY BOYD, M.B.Lond., F.R.C.S.,
Surgeon to the Hospital.

Cystic Adenoma of Ovary.

GENTLEMEN,—The first case is that of a girl, æt. 23, who was admitted for swelling of the abdomen, which she had noticed only since Christmas. There is a history of her having suffered from dyspepsia, and she has had various symptoms connected with anæmia, but they do not bear upon her present condition. The abdomen is very markedly distended. She used to measure 20 inches round the waist, but is now 34½ inches at that part. It is perhaps possible that all this increase may have come on since Christmas, but that would be unusually rapid. She presents all the signs of an ovarian cyst. The swelling rises from the pelvis, and you cannot press a hand backwards towards the spine above the pubes ; it reaches up to the costal margins. The swelling is perfectly uniform ; I have not detected any irregularity on the surface. It is distinctly outlined, tense, and elastic. Next, we find that the abdomen is markedly dull over the whole of its front until we reach the epigastrium. You will notice that she presents lesions (lineæ albicantes) due to clefts of the subcutaneous tissue. Yesterday I got a high-pitched tympanitic note

far back in the left loin, but am unable to elicit it to-day. The right loin is resonant. We therefore conclude that up to the line of resonance the abdomen is filled with a tumour. If we just flick the skin we get a well-marked wave right across the whole abdomen in all directions; therefore the swelling contains fluid. Examination *per rectum* shows the cervix to be over on the right side, the uterus probably being driven over with it. Towards the left broad ligament there appears to be a good deal of induration, which I believe to be due to some solid growth about the base of this tumour. Therefore our diagnosis is that there is an ovarian cyst, probably springing from the left side, consisting largely of one cyst with a mass of adenomatous growth or small cysts down towards the broad ligament. The operation for the removal of this tumour ought to be as simple as any that one can undertake. In this case there is no history of pain or anything to lead us to suspect the presence of any adhesions; but sometimes, without any warning history, there are adhesions over the anterior surface of these tumours, and one has to introduce the hand to separate the cyst from the abdominal wall; then on tapping the cyst the whole thing comes away as easily as possible.

Rapidly-growing Scirrhus of Breast.

This middle-aged woman is suffering from malignant disease of the breast. I have explained matters to her, therefore I can refer freely to the subject in her presence. It is the most rapid and worst case of malignant disease of the breast I have ever seen. Eight months ago she was knocked over by a bicycle, which caused bruising all down the side on which she fell, and there was apparently a good deal of swelling from subcutaneous hæmorrhage. She recovered from that completely, but four or five months ago she noticed that her breast began to swell as a whole, and at the same time she became aware of a lump in her armpit "like an egg." This she rubbed with some oil, when it seemed "to go up" behind the axillary fold. Three weeks ago her arm began to swell, and swelled rapidly down to the hand. Two or three weeks ago she began to have some difficulty in breathing. The pain has not been very great, and is of a sharp, shooting character, passing chiefly from the arm towards the breast. We know that lymph-œdema of the arm there is frequently

some interference with the brachial plexus, and that pain may be extreme. When she is lying quietly she breathes only twenty-one to the minute, but when she is talking there is distinct dyspnoea. She has a slight cough, with mucous expectoration, but no evidence of breaking down of malignant growths of the lung. The left chest is absolutely dull; vocal fremitus is absent, vocal resonance is good; a high-pitched to and fro breath-sound can be heard. The position of the heart apex cannot be determined, for the whole left side down to the iliac crest is markedly œdematous, a firm fold of swollen subcutaneous tissue overhanging the hip-bone. The contrast between the two sides is great; I have never seen such widespread œdema in a case of cancer of the breast. I think that the patient's dyspnoea is due to fluid in the left pleura; but as many of the physical signs might be produced by œdema of the side, I have asked my medical colleague to favour me with his opinion.

The whole breast is involved by the new growth and greatly enlarged. You can feel no tumour at all. The consistence is moderately firm. The skin is involved over the greater part of the breast, being much thickened, of leather-like firmness, with large gland orifices, and for the most part of deep purple-red colour. There is no tenderness; otherwise I was questioning whether it might not be a rare case of suppuration supervening upon malignant disease. At times such cases are extremely puzzling.

You will notice the distribution of the very numerous cancer plaques in the skin round about the breast. They are especially thick towards the mid-line, which they have crossed, and they are now involving the skin covering the left breast. Though doubtless due to carriage of cancer cells along lymphatic channels, they do not seem to follow the ordinary direction of the lymph flow; I believe this to be due to blocking of the ordinary lymph path, which necessitates the establishment of a collateral circulation along unusual paths, and thus epithelial cells are borne in unexpected directions. In this instance the whole axilla right up to the clavicle is full and firm, though no enlarged glands can be distinguished. Doubtless, most axillary lymphatics are plugged.

The next point in the case is that the breast is firmly fixed to the subjacent muscles, and probably to the thorax. I cannot feel the liver nor

any abdominal viscus. She has not lost very much flesh, and she complains of no bone pains.

Now, as to treatment—can anything be done for a case like this? Of course, removal is out of the question. But, as you may all have noticed, the operation of double oöphorectomy has been suggested and practised for cases of inoperable malignant disease of the breast. It has been practised also for malignant disease elsewhere, *e.g.* of uterus, but there has not yet been time to see its effect. The first case upon which double oöphorectomy was practised for inoperable cancer of the breast was under Dr. Beatson, of Glasgow. The case was as follows :—A woman had been operated upon twice for malignant disease of the breast. The clinical history, supported by a microscopic section, rendered

At the Clinical Society meeting last November, Mr. Pearce Gould showed a most remarkable case. The patient was a woman 43 years of age, who was admitted to the cancer ward of Middlesex Hospital with inoperable cancer. She had been twice operated upon, but again there was recurrence about the scar. There were also enlarged glands in both axillæ and in both supra-clavicular spaces; both her pleuræ were so full of fluid that she had absolute orthopnœa; moreover, her left thigh was shorter than the right by two inches, and was bent outwards, apparently as the result of a secondary growth in the upper third of the femur. She was absolutely confined to bed. When Mr. Gould first saw the patient he thought she was going to die very soon; but, though nothing supposed to be curative was done for her, she steadily



Rapidly growing cancer of left breast, involving the whole of the gland; skin over it generally infiltrated; œdema of left arm; infra- and supra-clavicular fulness.

the diagnosis of cancer certain. The surgeon who operated the second time declined to do so any more. She went to the Glasgow Cancer Hospital, and Dr. Beatson, who on theoretical grounds had come to the conclusion that removal of both ovaries might favorably affect malignant disease of the breast, obtained the woman's consent to the operation, after fully explaining the results to her and her husband. Several months later he showed her at the Edinburgh Medico-Chirurgical Society quite free from malignant disease, and later advices report the patient still well twenty-one months after operation.

Dr. Beatson at the same time showed a second case on which he had operated. The woman was improved but was not cured, and she has since died of the disease.

improved, and when I saw her eighteen months later at the Clinical Society, she appeared to be in ordinary health, and was walking about on this short leg,—lame because her thigh bone was still deformed in the way I have mentioned, but she could now bear her weight upon it. A little dullness of the bases of the pleuræ remained, supposed to be due to slight thickening. All cutaneous nodules but one, and all large glands had gone without any special treatment. On asking for the menstrual history it was found that at the age of forty-three, at a time when she was very ill indeed, she ceased to menstruate. Of course, there is nothing conclusive about these facts, but it is possible that some change had taken place in the ovaries which was practically equivalent to an oöphorectomy.

At the same meeting Mr. Bowlby quoted the case of a lady whose cancer recurred after a second operation, and for whom nothing further could be done. In her there was evidence of secondary growths in the liver. Without any treatment which could be regarded as specific, this lady's general health improved, superficial recurrences disappeared, and her liver shrank up again beneath her ribs. Nothing was stated as to the menstrual history; but I mention this case and the last rather to show that cancer may occasionally take a course with which I at least was not familiar—a course which would cause many to doubt whether the cases were really instances of cancer (though doubt appears to me to be impossible in these cases)—and a course which strongly encourages us to seek out its causes, and to endeavour to bring them into operation.

Dr. Beatson's writings, and his case above alluded to, suggest that the ovaries may exercise an influence upon the cancer process. There is not as yet much evidence one way or the other; but the following cases are worth mentioning. A well-known surgeon told me that a woman of about 35 with a large fixed mass in the axilla (secondary to a breast cancer which had been removed) consulted him, and at first he thought he would not be able to do anything for her. As she was leaving the ward his house surgeon said to him, "Why don't you remove the ovaries?" The matter was thereupon explained to the woman, and she readily consented to the operation. Five weeks afterwards the mass had become so much smaller and looser that, as the surgeon said, it might have been shelled out of the axilla.

I had myself at this time a patient on whom I had attempted a radical operation for the relief of recurrent growth, but had failed. The patient was still in good health, although she had suffered from cancer for four years. Her age was forty-five, and she was menstruating regularly. She had numerous nodules in the skin and subcutaneous tissue of pectoral region, one large mass in the great pectoral glands along the edge of the pectoralis minor, fulness and firmness above and below the clavicle and in the axilla. She heard of Dr. Beatson's case, and was anxious to have a double oöphorectomy performed. I put the matter as clearly as I could to her and to her husband, with the result that, though recognising the experimental

nature of the operation, they wished to have it done. I therefore removed both the ovaries on December 22nd, 1896. All pain disappeared at once. After a week there was distinct shrinkage of the affected parts and of most nodules. At the end of a month every one of the cancer nodules had diminished, and two of them had disappeared entirely. Two months after the oöphorectomy, on February 22nd, 1897, all nodules were either gone or much smaller. The large one in the great pectoral was the only considerable one left; and this, I am informed (March 27th) is still shrinking. General health is good.

Encouraged by this improvement, I operated (February 21st, 1897) similarly upon a woman of thirty-eight, who a year earlier had her right breast removed for cancer. She was very thin, and had a large number of nodules in skin and subcutaneous tissue, many being adherent to the chest wall. There was a large nodule in the clavicular portion, which alone remained of the great pectoral. General fulness of the axilla and one or two small glands to be felt. One enlarged gland lay above the clavicle. A considerable nodule in the opposite breast and one small gland in the axilla above it. A week later there was distinct evidence of shrinking of nodules and diminished vascularity of the cutaneous lesions. A month later she had gained flesh, her general condition had improved, all lesions had diminished, some had all but disappeared. The nodule in the left breast was markedly smaller, and the gland above it could no longer be felt.

These are all the facts that I am able to give you at present about this exceedingly interesting matter.

The tendency at first was to connect the effect of oöphorectomy upon the cancer-process with menstruation, and it was thought that, if a woman had ceased to menstruate, oöphorectomy would be useless. I think, however, that there can be no such connection, for we know that a woman may, and often does, develop cancer before, during, and after the menopause. But we all know that the ovary has a most potent internal secretion; for, if the ovaries be removed early in life, you do not get the full pelvic development or other sexual peculiarities. It seems to me that the best working hypothesis is that the internal secretion of the ovaries may become chemically altered, and may then favour the growth of cancer—to whatever cause

this may be due. If we remove a morbidly secreting ovary, the tissues may then be able to struggle successfully against the cancer.

To return to our patient. If this woman's chest will allow her to take an anæsthetic, I may feel justified in recommending oöphorectomy to her, as offering the only hope we can hold out to her.

[*Note*.—Nothing was done in this case, and the patient died not long after the lecture was delivered.]

Ileo-cæcal Intussusception at 8 months; Cæliotomy, Recovery.

This little girl, 8 months old, presents nothing for you to see or examine, yet she is very interesting, for she is an example of recovery after abdominal section for intussusception in a child less than one year old. The mother was getting out of an omnibus with the child when the attack began. It is conceivable that some sudden jerk started the intussusception, but the mother does not remember any jar; as she alighted the child threw up its arms, cried, and soon vomited the contents of its stomach. The illness continued, and the child became very pale. She was taken to St. Bartholomew's Hospital at 3.30 the same day; nothing wrong was detected, medicine was given, and the child was taken away. She remained very ill during the night, and refused the breast. When brought here the child had been vomiting for some six or eight hours; she continued to vomit after all food, pain persisted, and blood was passed *per rectum*, but otherwise the bowels did not act. That is an ordinary history of intussusception. On examination it was found to have a sausage-shaped swelling from the right side across the umbilicus to the left side. My house surgeon endeavoured to reduce the intussusception by means of water-pressure, but he soon reached a point beyond which he could not effect further reduction. When I examined the child I found that the swelling was confined to the right side, and lay rather above the level of the navel. I decided to operate at once. I made a median incision rather more above than below the umbilicus, which you will see the results of. I think the incision should be made in such a position as to enable you to get on to the swelling easily and quickly. I inserted two fingers, and was able to raise the intussusception to tilt it out through the wound, and then I tried to reduce

it in the ordinary way. I did not succeed at first, so I put my little finger in along the piece of bowel and gently dilated the opening and straightened the tube; then a further attempt enabled me to press out some five or six inches of considerably congested and thickened small intestine. The valve formed the apex of the intussusception. After the operation the child did very well indeed, and we ceased to anticipate trouble. But one morning soon after the operation the temperature ran up to 104° without obvious cause; the child, indeed, seemed so well that we were never very anxious about it. The temperature fell after three or four days, and progress once more set in, but we had another slight and short rise a few days later, also unaccounted for. The wound healed by first intention.

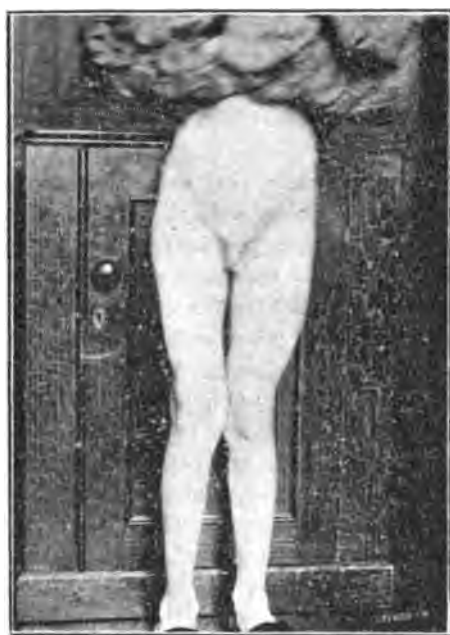
It is, unfortunately, very exceptional to get an intussusception doing well at this early age. I have done six or eight operations for intussusception, and have saved only the above child and one other. This was a boy who had a chronic intussusception, apparently of two months' standing; finally the bowel had protruded through the anus, forming a mass nearly the size of my fist. I reduced that under an anæsthetic; then I tried water-pressure and reduced it further, but beyond a certain point it would not go. As the child was nearly dead I had to suspend operations. The next morning, as he was rather better, I did a laparotomy, and the child recovered. This was my first case. The other children operated upon subsequently ought to have had a better chance, but I lost them one after another till this one.

In these cases circumstances connected with the patient have more to do with success or failure than have those conditions which the surgeon can control.

Sarcoma of right Femur and Tibia, apparently primary in each.

This boy, æt. 13, was admitted for a swelling of the right thigh-bone, and I show you photographs illustrating the state of affairs. The photographs show an excessive curving forwards and outwards of the upper part of the right thigh, with atrophy of the muscles, while the anterior superior spine of the right side is higher on the right side than on the left, which made him walk lame. There was nothing in the family history which threw light on

the matter. He is said to have been laid up with rheumatism three and a half years ago, after which he had a peculiar gait; but for one and a half years his legs and walk had been normal. Although the lad remembers having had vague pains in his thigh he did not take particular notice of anything until this New Year's Eve. Then he felt pain, and his parents noticed that he was beginning to get lame, and that the shape of his thigh was altering. When admitted the boy was very healthy-looking. The thigh could be felt



Sarcoma of right femur and tibia. Shows wasting of right thigh, abnormal prominence below trochanter, elevation of right knee and right anterior superior spine.

thickened in its upper third. As the fingers were passed upwards along the thigh the swelling seemed to end abruptly below the great trochanter. In the downward direction one could not feel any ending to the tumour. From before backwards the femur was certainly thickened. I next found that the right leg was an inch longer than the left—the right anterior spine being an inch higher than the left, as may be seen in the photograph. I next noted that the tibia on the same side was irregularly thickened and one inch longer than its fellow, thus accounting for the whole difference in length of the limbs. It is obvious, however, that the femur also was

lengthened, for though curved it was as long from end to end as its fellow. The condition of the tibia suggested congenital syphilis, but the boy showed no other signs of that disease, and there was nothing in the family history to point to it. We then got the boy up, and saw that as he got out of bed, or back into bed, he lifted his right thigh with his hands. This was strong evidence of some serious and deep lesion of the femur, rendering it unreliable as an organ of support in the limb—a point which was demonstrated still more clearly



Sarcoma of right femur and tibia. The side view shows marked bowing forwards of thigh in upper third.

when he walked. For although this did not seem to cause pain he limped badly, and in such a way as to convince one that he dare not bear his weight upon the limb. He heeled over towards the right (diseased) side in spite of the fact that the right anterior superior spine was higher than the left.

The diagnosis in this case was difficult. An examination of the femur alone strongly suggested sarcoma. The condition of the tibia seemed to be most easily accounted for by periostitis and osteitis. The femur, I thought, might possibly be the seat of a central gumma. Knowing that a sarcoma of bone was one of the most serious troubles that could befall the lad, I determined to cut down upon the femoral lesion, and try thus to solve the problem.

On reaching the apex of the tumour I found no infiltration of the tissues around the bone. The periosteum stripped easily, but was not thickened. With a chisel I now cut off a sort of apex to the bony swelling. The incision bled freely, and this gave me a strong hint that I was dealing with something serious. The piece removed by the chisel consisted of a cap of bone filled with a firm pinkish-white mass of sarcoma tissue. Permission having been obtained, I now proceeded to exarticulate at the hip-joint. Let me now draw your attention to the following points in the operation which I performed. One of the greatest difficulties in amputation at the hip is to control the hæmorrhage. An Esmarch's cord may be arranged round the thigh and pelvis, the loop round the thigh being prevented from slipping when the limb is removed by loops of bandage passed beneath it and drawn upon by an assistant; but the cord round the thigh may get in the way, and if it does slip a good deal of hæmorrhage occurs. I would rather trust to the fingers of a strong assistant compressing the external iliac at Poupart's ligament. On this occasion I took a well-curved handled needle, and passed it behind the vessels just below Poupart, making small cuts at the points where it entered and left the skin—partly to prevent the possibility of carrying any epidermis (and possible germs) down with my needle. I placed a small roll of bandage wrung out of lotion over the vessel, and tied a figure-8 ligature round it and the vessels, quickly and absolutely closing the latter. Previously I had applied an elastic bandage to the limb nearly up to the middle of the thigh. Almost the only blood lost at the operation was that contained in the femoral vein between the needle and the bandage. I paid no attention to the femoral vessels until I had secured all bleeding points in the posterior flap; then I picked up the visible branches of the femoral vessels, and when the needle was withdrawn I found that I had secured all.

The next most important point in a hip amputation is to keep the wound far distant from the perinæum—the most likely source of infection. Lastly, the operation should enable the surgeon to see the tissues he is dividing, so that he may be reasonably sure that he is not including in his flaps any dangerous material. Given these points, the more easily and quickly the operation can be per-

formed the better, but there is no need for excessive haste.

These points are well provided for in an amputation which I have taught and practised for some years. The patient lies on the back with the buttocks drawn to the edge of the table; the body is controlled and the sound limb kept out of the way by full flexion of this limb at both hip and knee. The femoral vessels are compressed as above recommended. A six-inch knife is entered through skin and deep fascia midway between the iliac crest and the tip behind of the great trochanter, and carried downwards just behind the line of the femur, whence it passes backwards for a short distance along the gluteal fold. Returning to the original line the knife is carried on till eight inches below the anterior spine, then it crosses the front



A case of large subperiosteal sarcoma of the femur, in which the method of amputating described in the text was employed.

of the thigh to the inner side, keeping parallel to Poupart's ligament, and an oblique cut is finally made across the back of the thigh from the end of this incision to join that in the gluteal fold. A short posterior flap is raised, including as little gluteus as possible. Then a skin flap at least four inches long is raised on the front and inner side of the thigh; next the muscles and femoral vessels are divided at right angles to their length, first the more superficial and then the deeper structures. As the flap is raised the hip-joint is exposed; the capsule is opened, the round ligament divided, the muscles attached to the great trochanter divided, the posterior layer of the capsule cut—the assistant aiding the surgeon by

powerful and steady extension of the limb, coupled with rotation out or in as occasion requires. Finally the knife is passed behind the head and the gluteus maximus, and the hamstrings are divided along the line of the base of the posterior skin-flap. The posterior flap is at once grasped to check bleeding, and the vessels are picked up one by one.

This patient immediately after the operation and for some days following did extremely well, and the loss of his limb did not cost him a tear. But after that he began to suffer from unaccountable pain in the stump. He is now getting well and picking up nicely. One other point worth mentioning about this subject is the value in such cases of the many-tailed bandage. Three overlapping pieces give a good hold on the body, whilst the tails surround and firmly compress the stump. Its application necessitates very much less lifting about. I am now using many-tailed bandages for many cases in which formerly I used the roller.

Let me now show you the bones from the amputated leg; they contain a great surprise. The



Sections of femur and tibia, showing large central sarcoma in shaft of each. No epiphysis is invaded. Both bones are bent, especially the femur. The dark patch in the femur shows where bit was chiselled out.

disease is a sarcoma of both tibia and femur, apparently primary in each. In each bone the disease consists of a mass of pink-white new growth occupying the medullary canal of the shaft, not showing

in either bone any evidence of having been built up of discrete nodules. If anything, the area of tumour is somewhat greater in the tibia than in the femur. As the new growth has eroded the more centrally situated bony tissue, fresh bone has been laid down upon the surface—at all points, so far. Thus has been produced the enlargement of the bones, whilst irregularity is due to the tendency of the growth to approach the surface more rapidly at some points than at others. This is best seen where the femur has become so weak that it has actually bent. Shortly, no doubt, the laying down of new bone would have been still less able to keep pace with growth; this would have fungated into the surrounding tissues, and a “spontaneous” fracture would have resulted. Whenever bone is subjected to excessive strain, nature always strives to meet the difficulty. In the femur in this case the natural buttress of the linea aspera in the concavity of the shaft of the femur is much exaggerated, and the femur is thus rendered very deep from before back. The result of examining the bones of the amputated limb makes me anxious, I confess, as to whether the boy has similar growths in other bones. I thought that there was some difference in size of these humeri of opposite sides, but the difference, if any, has not increased.

Recurrent Appendicitis; Operation, Recovery.

This next patient is a young man aged twenty-one, who has been operated upon for recurrent appendicitis. His first attack was eighteen months ago, the second twelve months ago, and the third three months ago. The first was the most severe. The history of this man is that he was at work as a newsagent carrying round papers, when he was seized with a severe pain in his right iliac fossa. It was so bad that he could not walk, so sat in a shop until a barrow was procured, upon which he was taken home. Before being put on to the barrow he was sick; but, later, vomiting was not a prominent symptom in the case. The pain continued in the right side, and the whole abdomen became rigid. The pain did not leave him for a month, but the rigidity subsided earlier, and then a lump was found in the right iliac fossa. His bowels were confined throughout the attack, though generally they are regular. Sometimes in these cases there is diarrhoea. I

think this occurs when there is pelvic peritonitis. The first attack laid him up for about three months. His second attack lasted six weeks, and the duration of the third was three weeks. The total loss of time from illness in eighteen months amounted to five months, a state of matters urgently requiring remedy. I did the ordinary operation, *i. e.* I made an incision close to the edge of the rectus abdominis, and came on to the cæcum, internal to which I found the vermiform appendix. I now show it to you. It is not more than an inch long, and has a swollen bulbous extremity; the whole wall is very much too thick. Near the cæcum the appendix is markedly constricted and the wall is thinned. The lumen is nowhere obliterated. The appendix contained a little mucus, no calculus or foreign body. I do not think there could have been very much real inflammation in these attacks, for there were no adhesions. Severe as the attacks were, they were probably due to appendix colic. I removed the appendix close to the cæcum by turning back, like a collar, a circular sero-muscular flap, tying a ligature round the exposed mucous membrane, cutting the mucosa through just beyond the ligature, and touching the stump freely with pure carbolic. Finally the sero-muscular flap was brought together over the mucous stump by a few stitches. The patient has had no untoward symptoms.

Notes on a Series of 115 Successful LATERAL LITHOTOMIES IN BOYS.

BY

FREDERICK C. BARKER, M.D., F.R.C.S.I.,
Brigade Surgeon Lieut.-Colonel I.M.S.; Medical Officer
to the Kathiawar Political Agency and in charge
West Hospital, Rajkote Kathiawar, India.

AFTER an experience of one hundred and fifteen consecutive lateral lithotomies, these notes are offered as an addendum to statistical tables like those of Mr. Gilbert Barling, writing in the 'British Medical Journal' of May 5th, 1894, p. 958, and other communications discussing the relative safety of the different stone operations. At p. 524 of the 'British Medical Journal' of March 9th, 1895,

Thompson's and Barling's tables show a mortality for lateral lithotomy in children under 12 and 10 years, of respectively 6 and 8·4 per cent.

While I do not suppose my happy experience is uncommon—the well-known immunity of children from the dangers of lateral lithotomy, doubtless furnishing many such records, or even more remarkable ones—I have nevertheless remembered from others that the operation is by no means void of danger, and have never approached it without the sense that accidents might arise.

The range of age in this series was from about one and a half years to twelve years. I may mention that under the age of fifteen years I have not, up to the present, lost a case in lateral lithotomy, though I could not have expected this at the outset of my experience, as I then found records of fourteen deaths out of 125 lithotomies (presumably lateral) in children, or 11·2 per cent. in a period of five and a half years.

In addition to the method of operating I have practised now for some ten years, and described in a communication to the 'Lancet' of 16th January, 1886, the main indications I always keep in view when operating are, first: *gentleness* with staff and forceps, neither blindly thrusting the one, nor putting two hands for traction to the other, even in adults, *a fortiori* in children. Extraction with an inadequate incision is resisted by the fasciæ, and levator ani in adults enough to decidedly hurt the operator's thumb. This is a good guide to what is going on in the deep parts, and (the stone being correctly grasped) *bistoury notchings* at the points of greatest obstruction are indicated. The application of two hands for traction to overcome resistance dangerously obscures the amount of force which is being applied. Proportionately greater caution is called for in children. I use the common probe-ended hollow-curved bistoury, slid flat along the stone held in the forceps, and turned on edge when and where the stone meets with resistance. I make a frequent practice of thus notching with a bistoury where necessary, though, of course, needlessly large incisions are as objectionable as insufficient incisions.

Secondly, never attempt insertion of the forceps merely *by rule*—driving them in a set direction instead of balancing them and lightly searching for the place of least resistance with their points

(dipped in castor oil) directed where a finger-tip has just gauged the hole made. With the forceps balanced between finger and thumb, and gently pressed against the aperture, practice soon teaches when insertion is to be obtained, usually occurring with a gratifying *slip*.

Thirdly, when exploring the rectum, if without counter-pressure above the pubes the stone is prominent, the patient being on his back, it is probable that the stone is large and bulges into the rectum simply by its weight, aided, perhaps, by a "hugging action" of the bladder. Even after long experience (here as in litholapaxy) flat stones are deceptive and may be extracted in their bad-axis after needless resistance, supplementary incisions and delay, unless a decisive exploration *per rectum* at time of seizure be undertaken. This may occur even with small stones; in case the seizure is difficult from size, the exploration and adjustment in the forceps are all the more important.

Before extraction is performed, the following shapes of stones should be kept in view: flat, cylindrical, globular, irregular, spicular, or *barbed*, and suitable measures taken.

I am in the habit of finishing the operation by flushing out not only the bladder and wound with boiled water, moderately hot, from a suspended pail, in a large stream through a glass nozzle, but also the whole colon till the water runs pure. The bowels are generally loose from the preliminary castor oil (preceded by a dose of *santonin*) and enema, and have not quite discharged all foetid contents, whose proximity to the cellular spaces it is well to prevent.

After the patient is put to bed, and while still under chloroform, tincture of opium with quinine is administered by rectum. This serves the three-fold purpose of bracing the system against infective accidents, of stopping the movements of the bowels for a time, and of quieting the patient.

I agree with Mr. Barling when he says ('Brit. Med. Journ.,' May 5th, 1894, p. 960) "that the operation which gives the best results at one age does not necessarily do so at another." With me, for adults, litholapaxy has nearly entirely superseded lateral lithotomy. Of course, I should deal with any huge stone supra-pubically, either, by Keith's (Hydrabad, Sind) splitting method, or by a giant lithotrite through the perineal incision and extrac-

tion in fragments, as in one case of my own—an old man—with a large *adamantine* stone, which, under the lithotrite, broke with an alarming report. He nevertheless made a singularly quick recovery, and was seen by me, seven years after quite well, though very aged.

TREATMENT OF CONVULSIONS IN INFANTS AND CHILDREN.

BY

J. MADISON TAYLOR, M.D.

CONVULSIONS from whatsoever cause require prompt and efficient treatment. They themselves may produce serious damage accompanied as they are by hyperæmia of the brain, more or less asphyxia, congestion of the lungs and of the various internal organs, thus throwing a great strain upon the heart, its valves, and upon the capillary vessels, and profoundly exciting the nerve centres. This tremendous disturbance may go on if unrelieved, and result in death. The general plan of treatment now to be outlined will do no harm, and probably relieve most of the commoner causes which induce the motor excitement. When a physician is called to attend a child in convulsions he should set out instantly. He should have ready to his hand certain articles enumerated below, or send for them while on his way to the case. The articles required are a bottle of chloroform, or ether, (or the mixture we use in whooping-cough, *amyl nitrite* 1 drachm, chloroform 3 drachms, ether 5 drachms;) also a bottle containing a solution of chloral, five grains to the drachm, a cylinder of oxygen, a hypodermic syringe, morphine, a soft rubber catheter, a fountain syringe, a clinical thermometer, a bath thermometer, and three or four rolls of cotton wool.

The child will usually be found already in a bath, which is often so hot as to injure the skin, producing sometimes quite serious results among panic-stricken folk. Take it out at once. If the bath contain mustard it may be so strong as to require rinsing off, and the application of some emolient, as petrolatum, and to wrap the child up in soft cotton wool, which in any event is a useful covering after the bath. If the child has not been

over-treated by domestic zeal and remedies, proceed to apply a hot pack at once, to which a little added mustard is useful, a teaspoonful to the quart of tepid water, 75° or 80°, in which dip a large bath towel or sheet and wrap around the child, covering it with a blanket, and leave it thus for from ten to twenty minutes. Meanwhile apply the chloroform or ether to the nose, and give a large injection of hot water, 95° or 100°, to the bowel. While the child is thus resting one can proceed to examine into the history of the case, and explore the lungs and heart, and when possible get specimens of urine and examine for albumin. Look into the nose or ears for foreign bodies, &c. Examine the penis for phimosis. The temperature should be taken also; if subnormal use stimulants. The hot injection acts well, helping to relieve the over-taxed heart to equalise the circulation, dilate the peripheral vessels, and also assists in unloading the bowels and intestines, or at any rate in liquefying the fæces. If the temperature be found high, the heat will relieve capillary congestion, relaxing the superficial vessels and expedite heat elimination. The hot injection can be followed later by cold injections, 70° or 55°. Also in hyperpyrexia, and indeed in most cases except shock, it is well to apply cold to the head. If the convulsion be very excessive or unduly prolonged, there need be no hesitation in using a hypodermic of morphia to a six-months-old baby $\frac{1}{8}$ grain, to a babe of one year old $\frac{1}{4}$, or two years $\frac{1}{2}$, and these may be repeated in an hour or two if no effect is gained. The second dose may be double the size of the first, for during convulsions the tolerance of opium is very great. Where there is asphyxia, oxygen is a valuable agent. Also, chloral may be given by the rectum in warm water or milk, four grains to a six months-old baby, six grains to one of a year, and repeated in an hour.

Gradually a knowledge can be acquired of what the definite cause may be, whether shock, fright, chill, heat exhaustion, sunstroke, congestion of the lungs, toxæmia, over-loaded intestines, the beginning of an exanthem, or other fever, or cerebral disease.

It is proper also in most cases to administer a purgative by the mouth, as calomel, or if the constipation be obstinate, croton oil, half a drop to a drop in glycerine or whisky. We have seen this save life when the cause was coarse, undigested

food partaken of three days previously, and the child had been abandoned to die. If the temperature remains excessively high, a cold pack may be used after the hot one, and whatever other remedies the nature of the case may require. A child who has had convulsions, especially if these exhibit a tendency to recur, must be kept absolutely quiet and fed in the simplest fashion, altogether by fluids, for several days. When the nature of the case is clearly revealed, one proceeds to treat that as required.

The Philadelphia Polyclinic, May 15th, 1897.

NOTES.

Resection of the Gasserian Ganglion for Rebellious Facial Neuralgia.—Marchant and Herbert ('Revue de Chirurgie,' April 10th, 1897, p. 286) report two cases of extirpation of the gasserian ganglion for the relief of rebellious facial neuralgia, and they analyse ninety-three additional cases collected from the literature. Among the whole number there were seventeen deaths (17·8) per cent. In sixty-six the temporal course was followed, with eleven deaths (12·12 per cent.), three of which were open to doubt. In twenty-nine the pterygoid course was followed, with six deaths (20·6 per cent.). Among fifteen cases of complete extirpation of the ganglion there were five deaths, three of which were open to doubt (13·13 per cent.). Among sixty cases of incomplete extirpation there were eight deaths (13·13 per cent.) Among fifteen cases of simple resection of the painful branches there was but a single death (a mortality of 6·66 per cent.). From a study of the literature of the subject the conclusion is reached that certain rebellious facial neuralgias originate in the gasserian ganglion, and the only treatment of these cases consists in destruction of the ganglion. When no appreciable lesion of the ganglion existed, and its removal was none the less followed by a disappearance of the neuralgia, this result is to be explained by the destruction of a nervous centre containing neurons or nervous cells whose prolongations only are affected by simple section of the nerve. The temporo-

sphenoidal course is the best to follow for the removal of the ganglion. The finding of one of its branches, and especially the inferior maxillary in the oval foramen constitutes one of the most certain guides for the detection and seizure of the ganglion. The ganglion may be completely extirpated. Commonly the extirpation is incomplete, and the ganglion is finally destroyed by curetting and crushing. Often only its branches have been resected. Hæmorrhage, wounding the nerve and cerebral compression are the immediate operative accidents to be feared. Secondly, there may be infection, hæmorrhage, iodoform intoxication, ocular disturbances, and otitis. As an immediate result of the operation there is a cessation of pain and also an abolition of general sensibility in the distribution of the three branches of the nerve, and especially in the second and third branches; but this does not persist long. Taste, smell, hearing and vision are variously affected. The movements of the jaw may remain impeded. From a therapeutic point of view recurrence is less to be feared after destruction of the ganglion than after simple resection of the branches. The evidence indicates that complete extirpation should be superior to simple destruction of the ganglion. *Journal of American Med. Assoc.*, May 15th, 1897.

Treatment of Tuberculosis of the Spine.—

Diakonoff ('Centralbl. f. Kinderheilk'nd,' 1896, i, 309) laid bare the seat of disease in three cases by resecting the vertebral ends of the ribs. Two of the cases were in a very poor condition, and died after the operation. The third case was described as follows: The child had a kyphosis in the neighbourhood of the seventh cervical and the first and second thoracic vertebræ, complete paralysis of the involved muscles, and motor disturbances of the lower extremities. Temperature was 99.9° F. The operation was done on December 17th, 1895. An incision 10 cm. long exposed the transverse processes of the second and third dorsal vertebræ. The first was cut through at its base, and resected along with the head of the second and third rib. In isolating the bodies of the vertebræ a pus cavity was discovered which communicated with the spinal canal through the foramina. After removal of the carious pieces of bone, partly with Lürer's forceps,

partly by the sharp spoon, the spinal canal was reached. The dura was found perfectly intact. The cavity was drained, the wound sewed up, and iodoform emulsion was injected through the drainage tubes. As the pus cavity very rapidly filled up with healthy granulations, the drainage tubes were removed on December 31st, and iodoform gauze strips were substituted. On the twenty-third day after the operation, motility was established in the paralyzed muscles, and at the end of January the patient was able to sit up for a long time, and carry the head erect without apparatus. On February 2nd he began to walk, and the motility increased.

Pediatrics, May 15th, 1897.

Peronine in the Treatment of Cough in Phthisis.—Dr. C. Schröder has compared the value of this salt—an hydrochloride of the benzyl-ether of morphine—with that of codeine phosphate in the treatment of obstinate phthisical cough, and concludes that it occupies an intermediate position between codeine and morphine. He used it in twelve cases in doses of from $\frac{1}{8}$ to $\frac{3}{8}$ grain (0.02 to 0.04 gramme). In eight cases it caused a notable diminution of the cough, in two cases larger doses were required, and in two others it failed completely. Prescribed in doses as large as $1\frac{1}{2}$ grains (0.08 gramme), however, it did no harm; in larger amounts it seemed to provoke nausea and have a decided constipating effect. In two cases sweating and difficulty of expectoration were noted; but in all the cases it produced a calmer sleep than that obtained with codeine.—*Gazette Hebdomadaire de Médecine et de Chirurgie*, February 14th, 1897.

Search for the Germ of Seborrhœic Eczema.

—Wm. H. Merrill, of Pepperell, Mass., gives the result of a series of experiments, and concludes that seborrhœic eczema is caused by a specific germ or germs, in form diplococci, whose life-history is most active at the ordinary temperatures and with free access to the air, but which can develop at much higher or lower temperatures and with a scarcity of oxygen,—a germ having the characteristics which one would most naturally expect in a disease as prevalent and widespread as seborrhœic eczema.

New York Medical Journal, March 6th, 1897.

THE CLINICAL JOURNAL.

WEDNESDAY, JUNE 9, 1897.

A Clinical Lecture and Demonstration ON THE PHYSICAL SIGNS OF CAVITATION OF THE LUNGS.

Delivered at the Brompton Hospital for Consumption
and Diseases of the Chest by

ROBERT MAGUIRE, M.D., F.R.C.P.,
Physician to the Hospital, and Physician to Out-patients,
St. Mary's Hospital.

GENTLEMEN,—I wish to speak to you to-day of the signs which lead one to find a pulmonary cavity, and of those which may lead one wrongly.

The detection of a cavity in the lungs is by no means a matter of mere expert curiosity, but is a real help for diagnosis and prognosis. By far the greater number of the cavities which exist in the lungs are caused by tuberculous change, and, as is well known, the signs of this are often doubtful, especially if the disease happen to be quiet. *A fortiori* then, if signs of a cavity can be found, our previously uncertain diagnosis is made certain. Further, probably no cavity smaller than a walnut can be diagnosed with certainty. It is true that at times one hears a physician speak of "diffused cavitation;" and post-mortem, it is true that in such cases small holes are sometimes found scattered widely through the lung tissue. These are caused by softening of caseating tuberculous masses, and the diagnosis of cavity which may have been made was based really upon the knowledge that such a process was going on, and that cavitation was probable. But no positive diagnosis of the presence of a cavity can be made unless the cavity has attained the size I have mentioned. It is almost certain that a cavity of such a size will never heal, at least that is what I myself think. The tuberculous process may quiet down and remain in that state for a long time. I show you a case in point which has puzzled many. Among my out-patients there was one about whom I used to amuse myself by asking the medical men present at my

clinic to say what they thought from appearance might be the matter with her. Usually they said that this was one of the ordinary dyspeptic cases with which one has to deal, and that there was nothing wrong with her lungs; yet that patient has cavities in most parts of her lungs, although she appears to be fairly well. So it is with another patient whom I show, a young girl.

The walls of a cavity may become fibrous and dry, and the patient may not suffer in any way from its being there, but the lung is not sound. In spite of the apparent well-being of the patient, some amount of caseous tuberculous material may rest buried in the walls of the cavity, may at any time infect the lymphatic or cardiac circulation, and lead to a fresh outbreak of the disease. Again, too, there may occur secretion from the wall of the cavity, a secretion which undoubtedly might contain tuberculous poison, and this may be insufflated into the bronchial tubes, and so may infect the lower parts of the lungs. The question of the spread of tuberculous disease in the lungs is one which has never been quite worked out; I shall therefore give you only my own ideas upon it. The lymph takes up the poison and spreads it by way of the lymphatic duct, the cardiac system spreads it by the veins and arteries, and the bronchial tubes are the means of drawing it into other parts of the lungs, and of thus producing secondary deposits of tubercle in the bases of the lungs. This is the cause of that peculiar form of tuberculous change which was figured by Carswell, of University College, and which is known as "Carswell's grapes," because of the grape-shaped appearance of the lesion. A greater danger often accompanies the presence of a cavity. It is in the walls of a cavity that aneurysm of an artery is prone to form, which may give rise to sudden, and nearly always fatal, hæmoptysis. During my term of office as Pathologist at this hospital I only once failed to find rupture of such an aneurysm in the cases of fatal hæmoptysis which were examined post-mortem, and usually, too, the aneurysms were found in only small cavities. Therefore, no patient with a cavity in his lungs can be said to be quite

safe, and thus the diagnosis of cavitation bears upon our prognosis. For these reasons I have wished to give you the physical signs which may be met with when cavities are present, and to point out those which, in my knowledge, are the most useful. I do so, also, because I find that students have great trouble with this subject, and are prone to lay most stress upon the least valuable signs.

It will be best to discuss the subject in the order of our usual course of examination of the chest, namely, under the heads of inspection, percussion, auscultation and voice signs.

Inspection.—The chest wall over a cavity is usually flattened, the result of the destructive process which has taken place. Yet it may be normal in appearance, or even more prominent than other parts where no cavity exists. Probably these exceptional signs are due to lack of rigidity of the chest walls—they will not give way,—or to adhesions which bind the lung to the parietal pleura. An undue convexity of the surface of the chest is found only when the walls of a cavity are unusually thin.

The movement of the chest wall over a cavity is generally lessened in extent, but this is not the result of the presence of the cavity, but of the pleural adhesions and of inelastic lung tissue which are most frequently its companions. Yet it is by no means unusual to find a normal extent of movement, especially in young subjects. The sign may be only the result of comparison with the opposite side, in which there is simple consolidation without such an expansile air-chamber as a cavity, and therefore a lessened extent of movement. But it often astonishes the inexperienced to find the more advanced mischief to be on the side of the chest, where, from the free movement, he expected perhaps only compensatory enlargement of the lung.

It will be seen, then, from what I have said, that inspection gives us no help for the detection of a cavity.

Percussion.—The percussion note over a cavity is a resultant of two conditions. The cavity itself ought to cause hyper-resonance, and when very large and with very thin walls it does so. But, nearly always, the cavity is separated from the chest wall by a layer of consolidated lung tissue, which should produce a dull percussion note.

Students at first do not understand why the note over a cavity should be in any way dull and not tympanitic, such as they expect from an air-containing chamber. But it must be remembered that the dullness is not due to the presence of the cavity, but to the thickened tissue of its walls. It is by this combination of tympanicity and dullness that we get the characteristic cavernous percussion note, which is of great importance and almost diagnostic. The note varies in its characters, according to the relative proportions of cavity and thickness of wall, and also is influenced by the presence or absence of a layer of healthy lung tissue between the cavity wall and the surface of the chest. It is best described as a note of high-pitched resonance, as was said by the late Dr. Walshe, and is almost identical in character with that which is obtained by percussion over the trachea when the mouth is open. For this reason great care should be taken, in investigating the apices of the lungs, to avoid a horizontal conduction of the tracheal note. The percussing finger should be directed downward and backward, without the slightest inward tendency, and here, as indeed for all parts of the chest, the slightest possible force should be employed. As with the tracheal note, the cavernous note is the more easily obtained and is the higher in pitch if the mouth be open, as Gerhardt has shown. Again, it is somewhat higher in pitch, also according to Gerhardt, during inspiration than during expiration; this, according to Friedreich, being caused by increased tension of the chest and of the cavity walls. Occasionally the note may disappear with a change of position, probably because of fluid which may block the communicating bronchus, though this is not likely to happen when the cavity is situated at the apex of the lung. A note similar to the cavernous note may, it is true, be got below the clavicle when a certain amount of fluid in the pleura has caused the lung to collapse to the upper parts of the chest,—I say advisedly not compressed but collapsed,—and, again, it may be obtained in rare cases of intense pneumonic consolidation by conduction from the large bronchial tubes. But these conditions are not likely to escape observation, and therefore I consider this queer percussion note as of great value in the detection of a cavity. In this hospital we call it the "box note," because of its curious characters.

The percussion note can also show not only the presence of a cavity, but also whether or no there be much fluid in it. The fluid naturally gravitates to the lowest part, and dulness may be detected in the sitting posture, which may be replaced by the high-pitched resonance when the patient lies down. Gerhardt, too, has pointed out how the shape of a cavity and the direction of its longer diameter may similarly be detected by percussion. Suppose that we have a cavity of an elongated shape one third full of fluid, and the patient is standing up, then the length of tube for vibration, as it is in an organ-pipe, will be less than that which obtains when the patient lies down, and the note obtained will be higher in pitch. That is what Gerhardt many years ago pointed out, and which was thought likely to be of value for diagnosis in consideration of the fact that it was once considered advisable to tap these cavities.

The so-called "cracked-pot" sound is of course to be obtained by percussion over a cavity. But apart from the fact that it is found in other conditions, I advise you to never attempt to obtain it. There is no need for it; any cavity large enough to produce it can easily be found by other means, and the forcible percussion which must be used to bring out this sign is not only very painful but also harmful to the patient. I show you a specimen of a cavity in the chest where a large artery has been laid bare, and you will here see the danger of rupturing the artery. I show you in this other specimen an aneurysm which has actually ruptured. You will see that before that aneurysm ruptured there was great danger of breakage from undue pressure, and here is a specimen of fusiform aneurysm in which the arteries are dilated and ready to burst. The specimen to which I wish most to draw your attention is this in which you see an artery crossing a cavity, unsupported and ready to burst at any moment. Now, if you remember that according to the teaching of the schools one of the conditions required to enable you to hear the cracked-pot sound is that you must percuss heavily, you will understand the risk of this proceeding. The cracked-pot sound is heard sometimes over rickety chests, and, for this simple reason, that the chest gives way to your heavy percussion, you knock the wind out of the lungs for the time being, and so produce the cracked-pot sound. Again, it is heard over pneumonic consolidation, and that I cannot

explain. If you put one finger on the table, and leave a little air under it, you can produce a cracked-pot sound by hitting the finger with another finger. The sound can be produced by clapping your clasped hands together with air between the palms. Now imagine what that percussion might do over a cavity in the chest when there may be an aneurysm ready to burst. The chest should always be percussed lightly, as I have elsewhere shown.

Auscultation.—The classic sign of a cavity is "cavernous breathing," and this is the most reliable of all the signs, if only the observer can be certain of its presence. Unfortunately it is not always present, even though the state which may cause it be there. It varies greatly, too, in its character, and may be so imperfectly marked as to be indistinguishable from ordinary bronchial breathing. Its essential character is hollowness, and when produced by a large cavity it is almost exactly the sound got by blowing over the mouth of an empty bottle. But its pitch varies with the size of the cavity. When this is small the tone got by percussion may be so high as to resemble closely the tubular breathing which is heard over pneumonic consolidation. The difficulty of distinguishing between cavernous and bronchial breathing may be sometimes overcome, according to my colleague Dr. Reginald Thompson, by an observation of the pitch of the inspiratory and expiratory sounds respectively. In bronchial breathing the expiratory sound is usually higher in pitch, and in cavernous breathing lower in pitch than the inspiratory sound. But such is by no means always the case, and the distinction must not be too much relied upon. In fact it is impossible to convey to another accurately in words the characters of cavernous breathing. The only way in which a student can get to know them is by having the sound pointed out to him and then listening until he hears for himself its peculiarities. I think that I can, in a certain way, describe the differences of these sounds. The bronchial sound is what you hear yourself when you put your throat into the position to utter the German "ch," and then inspire and expire. It is a guttural sound, and you will find the expiratory apparently higher in pitch than the inspiratory sound. The tubular breathing, such as you hear in pneumonia, is that which you notice when you respire with the tongue

raised to the hard palate. The cavernous breathing is heard by yourself if you respire forcibly, placing your mouth in the position for whistling, but with the lips a little more open than needful for that amusement. The sound is best heard over a cavity which has hard dry walls and contains but little fluid, in fact the more fluid the cavity contains the less marked is the cavernous breathing, and this explains to some extent why the sound disappears at times over certain spots and yet may again be heard over the same spot.

I wish to refer here to a case I had amongst the out-patients when I was in charge of them, which showed how one may be deceived by physical signs. A man came who had a typical phthisical aspect, and unquestionable tubercular disease of the larynx, but I found nothing wrong in the chest. That man came three times with an interval of fourteen days between each interview, and still nothing wrong could I find in his chest, but on the fourth visit we found obvious cavities in all parts of his lungs. The cavities had been there, but they had been blocked, and it was impossible to diagnose them in that condition. It is not necessary here to discuss at length the mode of production of the sound. Probably it is in most cases not due to air entering and leaving the cavity, for it may be heard when there is absolutely no expansion of the chest, as in large old cavities and also in pneumothorax. Again, the sound is heard throughout the whole of both inspiration and expiration, and it is impossible for a cavity of long standing to expand and contract sufficiently freely to produce this by its own walls. It can be heard when the diaphragm is tied down and prevented from moving by tubercular peritonitis. The most likely explanation is that the ordinary so-called "bronchial sound," produced near the cavity resonates or echoes in the empty chamber. It has been said to be possible, too, that consonation may play a part, cavities of different sizes consonating with breath sounds of similar pitch. Here, however, I must guard myself against being understood to admit the theory of consonance for chest sounds, and particularly those of the voice. Though I have so great an authority as Skoda against me, I think that consonance plays no part in the causation of voice or breath sounds in the chest. I have elsewhere explained my reasons for this view.

In Gerhardt's text-book you will find described how Helmholtz's resonators may be used to increase the sound caused by percussion over pulmonary cavities. This is interesting, but limited in its application, and of no practical utility.

The rattles produced in the neighbourhood of a cavity, or even in the cavity itself, by disturbance of mucus or pus, acquire peculiar characters. They are always metallic in sound, as is heard when tuberculous nodules are softening, and it is from the detection of such metallic rattles that we hazard the diagnosis of diffused cavitation already mentioned. But the rattles also assume a "hollow" character, if they are produced near or in a cavity of reasonable size, and then, as so-called "cavernous râles," they form a reliable guide to the detection of the cavity. Their peculiar sound is also produced by resonance or echo. Again, an exaggeration of the cavernous râle, the "splashing râle," may be heard over a cavity containing fluid, when the patient coughs, and so disturbs the fluid. This sign too is valuable, and was, I think, first mentioned by my colleague Dr. Mitchell Bruce, as the "tussive splash."

I may refer here also to "succussion splash," which is heard in hydro-pneumothorax, when the patient's body is shaken to and fro. The same phenomenon may be observed over large cavities containing some fluid, but as such cavities must be exceedingly large, they can be easily detected by other means, and this sign, therefore, is not needed.

Cough produces three sounds from a cavity; one is the sound to which I have just alluded, the "tussive splash," another is "tussive resonance." If you listen over the healthy apex of a lung the sound of a patient's cough is not reinforced; but if a cavity is there, you hear what is called "tussive resonance," which may be even painful to the ear in its intensity. The third sound is a very peculiar one, and requires considerable practice to detect. It is the "post-tussive suction," or the "india-rubber ball" sound. When a hollow perforated india-rubber ball is squeezed, and then allowed to expand suddenly, a sound of suction is of course heard as the air re-enters the ball. So with a cavity; when it has been compressed by the expiratory effort of a cough and is then suddenly released, its walls expand from relief of the pressure upon them, and a certain amount of air re-enters the

cavity. This produces a suction sound, and with it, too, are generally heard crepitations, produced probably by the movement of the hard walls of the cavity. The suction sound must, however, be carefully distinguished from the inspiratory sound which naturally follows a cough. It precedes this, and a little care will avoid the error.

I have spoken about the danger of rupturing pulmonary vessels, "lung vessels," we will call them for the present, by percussing too hardily, and thus depressing the chest walls over the cavity; so with cough, there is pressure on the cavity and danger of rupture. We have two arteries in the lungs, one bronchial from the aorta with blood under high pressure, and the nutrient artery of the lungs; the other the pulmonary artery from the right ventricle with blood under low pressure. This has thin walls, and is not a nutrient artery. We are told, and it is probably true, but I think it has never been proved, that the early hæmorrhages in phthisis come from the bronchial arteries supplying the smaller air-tubes. But we have hæmorrhages occurring without phthisis in Bright's disease and high arterial tension; most likely these come from the bronchial arteries. Again, we have the hæmorrhages in late phthisis; I do not know, but probably these are from the pulmonary artery, which is under low tension, and the blood may be sucked out from them by this expansion after cough which I have mentioned. That I think is the cause of the presence of pulmonary aneurysms. For some one with time for the work it would be worth while to inject the bronchial and pulmonary arteries respectively in an advanced case of phthisis, and with different coloured fluids, so as to determine the origin of these aneurysms.

There are two other sounds which may be heard over very large cavities; metallic tinkling from the explosion of bubbles of air in the fluid contained in the cavity, and the bell-sound, heard when a coin placed over a cavity is struck by another coin. These, like some other phenomena mentioned above, are present only in advanced cases, when their presence is of no help to the physician.

The heart-sounds occasionally resound in a neighbouring cavity, and the beat of the heart against the cavity walls may produce a systolic murmur by driving air out of the chamber. Similarly, too, I have heard the heart-beat produce

splashing râles at each systole in a neighbouring cavity containing fluid; but these sounds are found in other conditions, and are of no value for diagnosis.

Voice phenomena.—Vocal fremitus is usually increased over a cavity, but it may be diminished if there be a great thickness of pleura, the result of chronic inflammation.

Vocal resonance is increased, both in the form of bronchophony and of whispering pectoriloquy. But I cannot too strongly insist upon the almost absolute worthlessness of these signs in the detection of a cavity. It happens to me frequently to find that a student diagnoses a cavity because he has observed dulness and whispering pectoriloquy. These tell nothing of value, and whispering pectoriloquy is certainly the least valuable of all the signs to be observed over a pulmonary cavity. It is to be heard well over pneumonic consolidation, sometimes over an empyema in a child, and I have heard it best of all in a case where an abscess between the liver and the diaphragm had pushed the lung upwards. It may be added to the other signs in doubtful cases, but on no account should it be greatly relied upon.

To summarise, then, the signs I consider to be of most value in the detection of a cavity are—(1) the cavernous percussion note; (2) cavernous breathing; (3) cavernous râles and splashing râles and tussive splash; (4) tussive resonance; and (5) post-tussive suction.

But no one of these signs alone should be depended upon; an accurate diagnosis can only be made by combining the observations of them all.

Influence of Concomitant Disorders on the Diphtheria Bacillus.—Faber states that after diphtheria the bacilli successively become scarcer during convalescence; but when a new disease—for instance, scarlatina—sets in, the bacilli of diphtheria often disappear suddenly. As diphtheria may complicate scarlatina, there can be no antagonism between the specific microbes of these two diseases, but probably the sudden disappearance of the diphtheria bacillus is due to an antagonistic effect of the streptococci which so commonly are found in the throat in scarlatina.—*Hospitals-Tidende*, p. 1083, 1896.

PROSTATIC DISCHARGES.

Their Causes, Symptoms, Diagnosis, and Treatment.

BY

CAMPBELL WILLIAMS, F.R.C.S.

PROSTATIC discharges, from the frequency of their occurrence and persistency, together with the annoyance they entail on patient and practitioner alike, have gained for themselves an unenviable notoriety. They arise from diverse conditions and causes, and may be "infective" or non-infective. The discharge may be mucoid, muco-purulent, or purulent in character. The amount of secretion varies from shreds or dampness up to a free discharge.

The condition may arise from :

1. Simple hyperæmia of the prostatic mucous membrane.
2. Granulations superadded to the hyperæmia.
3. Parenchymatous prostatitis in addition to superficial hyperæmia.
4. Hyperæmia, granulations and parenchymatous mischief combined.
5. Inflammation of the vesiculæ seminales— α , acute ; β , subacute ; γ , chronic.
6. Tuberculous deposit or ulceration.
7. Polypi— α , mucoid ; β , fibroid.
8. Prostatic abscess ; superficial, deep.
9. Prostatic calculi.

The causes may be classified as predisposing and exciting. In the first category are found gout, anæmia, varicocele, congenitally narrow meatus urinarius, and sedentary occupations.

As exciting, one must enumerate :

1. Excessive venery, normal or abnormal.
2. Gonorrhœa which has extended posteriorly.
3. Gouty inflammation.
4. Phosphuria and lithæmia.
5. Rectal irritation, piles, ulcers, fissure or fistula.
6. Tubercle of the prostate.
7. Organic stricture.

The symptoms of prostatic discharge can be divided into objective and subjective.

1. Simple hyperæmia of the prostatic mucous membrane.

This is usually sequent to sexual irritation or excess, but sometimes to phosphuria or lithæmia, which act as mechanical irritants. When hyperæmia is present in a very slight degree, the patient may be unaware that anything is abnormal until he discovers a clear stain upon his linen or notices a dampness upon his person. When the amount of congestion is greater he has a constant clear glycerine-like discharge, which is increased at stool from the action of the levator prostatae. The meatus is often glued up so that the stream of urine is twisted at starting. In many cases the act of micturition is increased in frequency, and is accompanied by slight warmth at the neck of the bladder. But pains, local or referred, are absent. Many complain of "tickling or pricking sensations" in the urethra difficult to localise. On gently squeezing the corpus spongiosum between the fingers this symptom can be elicited if absent, or intensified if present. The irritation may be referred to the anus. So marked is this in some cases that the patient cannot refrain from scratching the parts. Some complain that the penis feels cold and has shrunk. On inspection the surface of the glans penis may have a crinkled aspect, whilst a bluish pink zone may often be seen around the meatal orifice. The inner surfaces of the meati have a bluish congested glistening appearance and are moister than normal. The sexual orgasm is too quickly effected, and a sense of heat during or after its occurrence is noted. The seminal fluid emitted is increased in amount and is of thin consistency. The power of repeating the sexual act is frequently long delayed. The patient experiences a feeling of great prostration. Coitus is often followed by an immediate temporary cessation of the discharge. But it usually reappears in from three to five days time. I have noticed that after connection the frequency of micturition is increased, but passes off with the return of the discharge. The contact of cold water with the hands seems in these cases to produce an inordinate desire to urinate, which is restrained with very great difficulty.

Mental distress on account of their condition is marked. Many are almost hypochondriacal on the subject of their ailment. They are often the victims of masturbation. But I have met with this phase in the bridegroom after hymeneal intemperance. Let me emphasise that the term

"intemperance" is only one of degree depending on the idiosyncrasy of the patient. Some people require but a very slight amount of sexual irritation to produce prostatic hyperæmia, the foundation having been laid in their schoolboy days. In all my cases I have noticed that the urethra is highly hyperæsthetic; and that they are liable to frequent nocturnal emissions. The subjects of prostatic hyperæmia are usually habitually constipated, anæmic, dyspeptic, and sedentary, whilst the lesser degrees of varicocele or hæmorrhoids are present so frequently, that one is inclined to exclude coincidence.

In cases of simple hyperæmia I do not examine endoscopically, but where it has been done I have noticed the following conditions:

Assuming that the meatus is of normal size, no stricture being present, the tube of an endoscope can be passed with ease, from the surgeon's point of view, until the region of the prostate is reached. Hyperæsthesia is so marked in these cases that it is advisable to use a 5 per cent. solution of cocaine to facilitate examination. Inspection of the anterior or penile urethra reveals practically nothing. But when the prostatic portion is reached, slight obstruction to its passage is encountered. There usually is an outcry as soon as the triangular ligament is stretched, and contact of the tube with the verumontanum produces pain, burning, and a violent desire to micturate. The lubricant and mucus having been mopped up with wool upon suitable holders, the prostatic mucous membrane comes into view. The colour in health is a full pink, taking the penile urethra as pink. It has always struck me that necessary depression of the tube to reach the prostate may from pressure produce a certain increase of colour. That the difference between the penile and prostatic tints would not be so widely marked were it possible to examine them without the aid of instruments. In simple hyperæmia the hue is brightened to crimson, cardinal red, or bluish red. The delicate striated lines on the surface of the caput are either very faint or invisible. The crescentic shape of the prostatic urethra is well marked.

Treatment.—In cases of this category I refrain from intra-urethral treatment. As a rule they get well by simple methods. I am sure that there is a liability to set up parenchymatous mischief from using endoscopic tubes and steel sounds. Injec-

tions do no good, if they do not even make them worse. In many cases they undoubtedly produce or increase irritation. One should primarily endeavour to secure rest, mental and local, from sexual excitement. In some cases, however, too long continence is worse than intercourse. This is owing to frequent recurring priapism. Moderate intercourse relieves the congestion. Urinary defects should be rectified. Thus in phosphuria, acid. nitro-hydrochlor. dil. with ext. parietæ liq., and decoct. tritici repens, will correct that condition. Where excessive acidity or urates are present, alkalies and hyoscyamus fulfil the requirements. Sandal-wood oil seem, in my experiences, to make these cases worse. Constipation must be overcome without resorting to violent purgatives. A very useful pill, almost homœopathic in its dosage, is the following:—Calomel, aloin, ext. coloc. co., ext. hyoscyamus, āā gr. $\frac{1}{2}$, ext. gent. gr. j.

Anæmia is combated best with the tinct. ferri sesquichlor. \mathfrak{m} 15 to 30 t.d.s. Nocturnal emissions can be controlled by ext. salix nigra liq. in full doses. This does not depress or produce acne like the bromides, or camphor grs. ij, ext. belladonnæ gr. $\frac{1}{8}$ in pill for a bedtime, as in chordee, is useful as an erotic sedative. The usual routine treatment of emptying the bladder, light evening meal, cold douche, and hard cool bed should be enjoined. If hæmorrhoids are present a suppository of hamamelidin grs. ij, ext. belladonnæ gr. $\frac{1}{8}$, pulv. trag. and but. cacao at bedtime give relief and rest. Exercise is good, whilst change of air to a bracing climate often effects a cure where medicine fails. Alcohol in small amount does not seem to make them worse.

Granulations superadded to hyperæmia.—These are most frequently a sequel to gonorrhœa, but may occur from repeated attacks of simple hyperæmia. When due to the first cause, their infective possibilities and their bearing on the marriageable state have to be considered. This condition may be suspected from the persistency and character of the discharge. It is this class of case that recurs, for it seldom gets well without special treatment. The "visible" discharge may cease, and the patient thinks he is well except that his prostate never feels quite right. If at this stage one examines the urine in a glass, it will be found to contain numerous purulent and mucous shreds. Sexual excitement or, perchance, a drinking bout

will convert this into a perceptible discharge. The patient bemoans the fact that he has caught yet another gonorrhœa. Such is not the case. He has only lighted up into activity the mischief that was present all the time. When of visible amount, the discharge may be mucopurulent or mucoid by day, and thickly purulent on waking in the morning. It seems to get concentrated in the urethra overnight. I have noticed that when the patient squeezes up discharge for the surgeon's inspection, that he often produces a bead of pus surrounded by clear glycerine mucoid-like discharge, very similar to the yolk of an egg suspended in the white. One meets so many of these cases that *are free from all referred pains* that one is forced to conclude that a certain amount of parenchymatous prostatitis is *requisite* for the production of the latter. Should, however, the granulations be situated in the immediate vicinity of the orifices of the seminal ejaculatory ducts the patient experiences a sharp, hot, burning pain on emission. So much so, that they may be forced to bathe the perinæum to obtain relief. This burning is usually accompanied by painful and frequent micturition. Drops of urine are passed every few minutes, and the passage thereof is described as if molten lead were being voided. It may last from 15 to 30 minutes.

On examining the urethra with an endoscope, it is usual to find granulations in the penile portion, unless they have been previously cured. On reaching the prostatic region, the view is usually obscured by blood—the granulations having been bruised by the passage of the tube. Having mopped this up and checked further flow by suitable styptics, such as hazelene, iron, or pressure, the granulations will be seen either in groups or isolated. They are plainly visible as dark red dots on the surface of the mucous membrane.

Treatment.—Endoscopic treatment is of the greatest value. It is immeasurably superior to installations of nitrate of silver by means of either Guyon's or Ultzmann's syringes. One is able to paint the individual granulations through the tube without irritating that portion of the mucous membrane that does not require treatment. The method employed is, after having cocainized the urethra, to pass an endoscopic tube to the prostatic portion, and to paint the granulations that come into view with solutions of nitrate of silver. Usually,

nothing under 20 grs. to the ounce of distilled water seems efficacious. As a rule I use a 10 per cent. solution when granulations are well marked. Great distress after the effect of cocaine has passed off is common. Intense dysuria and strangury can be obviated in a degree by emptying the bladder of urine, and throwing in six to eight ounces of warm boracic lotion *prior* to the painting. If much pain is present, a very hot hip-bath gives relief. In one very intractable case, the patient was put under gas and ether, and the prostatic urethra mopped out with a 30 per cent. solution of nitrate of silver. It completely cured him. Such heroic measures are seldom required. Solutions of sulphate of copper, 15 grs. to the ounce, or even blue stone, on a suitable holder, can be used to treat the granulations. When painting has been effected, the following mixture is a good urinary sedative, and eases their painful urination: potass. cit. grs. 20 tinct. hyoscyamus \mathfrak{m} 30, decoct. hordei \mathfrak{z} j, t. d. s. It is well to warn the patient that he may expect bleeding at the end of micturition for a day or so following treatment. They often pass several drops of pure blood at the end of the act, and would feel nervous if not warned about the liability of its occurrence.

Parenchymatous prostatitis in addition to superficial hyperæmia.—This is sometimes of gouty origin, or may be set up by instrumentation, but it is most frequently the result of gonorrhœa. In this condition a purulent discharge is, as a rule, continuously present, it may be scanty during the day, and copious, thick and creamy on rising. There is a sense of fulness, bearing down, or even tenderness in the perinæum. The apex of the prostate can be felt very plainly, and digital examination per rectum is painful. The prostate is tender, full, soft, and boggy to the touch. The enlargement is usually more marked on one side than the other, in fact *it*, together with the tenderness, may appear unilateral. The stream of urine is diminished in size. The amount of diminution may vary from day to day, apparently owing to the degree of swelling of the verumontanum. This seems to act as a ball-plug. There is increased frequency of micturition, together with scalding. There is an after-dribble of urine at what should be the end of the act. In addition to local discomfort, the patient suffers from referred pains. These keep his mind dwelling on his condition, and make him

morbid. Exercise causes an exacerbation of his aching, and he becomes inordinately sedentary. For, although rest in bed is enjoined in the acuter conditions, the chronic cases are improved by moderate exercise. The referred pains have numerous sites. According to Head, they have a distinct bearing on that portion of the prostate affected. They may be in the lumbar region, so renal disorder is suspected. With care, the definite point can be ascertained, but as a rule the patient has difficulty in exactly locating the spot. It may be constant or intermittent. It may vary from a dull, stiff, aching feeling in the small of the back, most marked on waking, to an amount that incapacitates the patient for exercise, or from following his daily vocation. Other sites are suprapubic, usually ascribed to acid cystitis. In the inguinal regions, the lymphatic glands are blamed—wrongly. The sacro-iliac synchondrosis, hip-joint, coccyx, great sciatic nerve, inner side of the thigh, calf of the leg, the ball of the great toe are other regions to which the pain is referred by the patient. The supra-pubic and inguinal sites are the most common in my experience. One or more of these pains may be complained of at the same time, or the locality may vary from day to day. These cases do not seem to experience discomfort at the corona of the glans penis, as in the hypertrophied prostate. The pain is described as a dull aching, and is increased if they allow their mind to dwell upon it. If their attention is drawn from their condition by congenial occupation, it frequently disappears, only to return in leisure moments. Prostatic abscess is an occasional complication that may occur in these patients. Repeated or continued attacks may lead to atrophy of the verumontanum.

Treatment.—In the acute cases rest in bed is to be enjoined, but moderate walking exercise in the sub-acute or chronic conditions. The bowels should be kept gently open by confection of senna or cascara or other suitable methods. Very hot hip-baths, in which the patient should sit for 15 minutes, should be employed twice daily. Throwing 4 to 6 ounces of cold or iced water into the rectum night and morning gives relief. Some patients experience better results from using hot water injections. They should be retained for at least 5 to 10 minutes. At first this is difficult for the sufferer to manage, but he soon trains himself

to it. The benefit attained by a perineal blister is, to my mind, discounted by its after discomfort. Its employment has often lost a practitioner his patient. Painting the prostatic mucous membrane with a solution of nitrate of silver (10 grs. ad 1 oz. of distilled water) has given relief in many cases from the referred pains. This freedom may last a week or more. *In the gouty form ALL treatment per urethra does harm.* But colchicum, in full doses, in combination with alkalies is efficacious in controlling the pain and checking the discharge. A suppository composed of ext. belladonna gr. $\frac{1}{8}$ to $\frac{1}{4}$ and morphia gr. $\frac{1}{4}$, used night and morning, is very useful and sedative. Hot glycerine and belladonna fomentations to the perinæum are comforting in the acuter forms. In the non-gouty condition, alkalies, Bojeau's ergot, camphor, belladonna, hyoscyamus, antimony, copaiba, &c., are all useful drugs. All injections when used by the ordinary urethral syringe seem, in my experience, to be productive of harm rather than good.

Hyperæmia, granulations, and parenchymatous mischief combined.—I have invariably found this combination to be the after-result of gonorrhœa, and consider it to be infective. It is seldom that the trouble is confined entirely to the prostatic urethra, but one often sees patients in whom the penile portion has been cured whilst the posterior part has not been treated. Local application is a necessary and certain method of treatment in this condition. It is needless to say that should a granular state be discovered in the prostate, that one ought to carefully examine the penile portion in its entire length, to exclude similar mischief. In so doing, one is apt to find slate coloured patches—the remains of former inflammatory trouble. Care is requisite not to confuse this condition with the shadows thrown from the endoscopic tube. When they, the patches, really exist, no movement of the tube or light barrel will obliterate them, whereas the reverse is the case with shadows. Sometimes circumscribed, discrete patches of congestion are met with. As this article is more particularly devoted to the prostate portion of the urethra, I will refrain from any mention of the numerous divergencies from the normal that may be met with in the penile urethra.

The treatment here consists of topical applications to the granulations. Should the penile

portion be affected, an injection of zinci sulph. grs. iv, alum sulph. grs. iv, cupri sulph. gr. $\frac{1}{3}$, ferri sulph. gr. j to \mathfrak{z} j, aq. dest., *diluted with an equal part of hot water* will be found useful. But it will not do good to the posterior urethra. This is, of course, combined with suitable mouth treatment as advocated in allied conditions.

Inflammation of the vesiculæ seminales.—This may be of the acute, subacute or chronic variety. Excluding tubercle, which is usually an extension upwards from the testicle along the vas deferens, one finds this condition to be due to inflammation that has spread downwards from the sinus pocularis, *viâ* the ejaculatory duct, and which has set up vesiculitis. Its origin can be traced as a rule to gonorrhœa. Its presence may be suspected from a discharge more or less continuous at one time, with an apparent cessation at another. This latter phenomenon being due to natural or intentional emptying of the vesicle. Nature effects this end when the patient strains at stool. He notices a sudden flow of pus, and his discharge then ceases for a day or so, only to return after it has collected again in sufficient amount. The surgeon can attain a similar result by pressure on the prostate. One hand is placed over the pubes, whilst the finger of the other is passed into the rectum. Extrusion of the contents of the seminal reservoir is made by approximation of the hands. In these cases a seminal emission is of diagnostic value. In the acute form pure blood may be present. In the subacute and chronic the emitted fluid will be found to be of a brownish-red colour from the presence of altered blood. The patient frequently starts up from his sleep through the pain caused by the orgasm. The passage of a hard motion causes a dull aching pain in the rectum, usually *unilateral*. I would here remark that, in my experience, it is common to find only one vesicle affected rather than a bilateral condition.

Patients have complained that pain caused by defæcation has shot "down their sciatic nerve" *on the same side* as the affected portion. The vesicle can be felt, *per rectum*, to be enlarged and sacculated, and is exquisitely tender. Pressure causes extrusion. The fluid may be pus or a thin brownish-red fluid. Microscopically one finds red blood corpuscles, pus, and epithelial cells, together with spermatozoa—usually dead. The gonococcus may be discovered if the fluid is stained. Suppura-

tion of the vesicle may take place through the ejaculatory duct becoming blocked, and the contents may be discharged into the rectum.

Endoscopic examination may reveal nothing, but I have seen pus well up into the sinus pocularis, which confirmed the diagnosis.

Treatment of these cases requires perseverance on the part of the patient as well as the doctor, for it consists of regularly and periodically expressing the vesicle. But even this seems unavailing in some cases, so much so that it has occurred to me to treat them surgically by incision and drainage as the most probable method to effect a cure. Though injections cannot have any curative power, as the seat of mischief is beyond their reach, they serve to keep the urethra clean.

Permanganate of zinc (gr. $\frac{1}{3}$ to \mathfrak{z} j distilled water) or hydrarg. perchloridi 1 in 10,000 attains this end. Some urethræ are singularly intolerant of the mercurial solutions, even when as dilute as the above, but a saturated solution of boracic acid, used with an equal part of hot water, is well borne. The heat relaxes the muscles, and allows the injection to get well back.

The rectal plug is an instrument which is worn to keep up pressure on the prostate. I have tried it, but was not impressed by its use. It caused great discomfort to the patient without an adequate beneficial result to the discharge.

Tuberculous deposit or ulceration.—Tuberculous mischief starting in and remaining confined to the prostate does occur, but is extremely rare. But disease in this gland, in conjunction with the manifestations elsewhere, is not uncommon. The trouble may be apparently limited to a single tuberculous focus, or there may be multiple foci disseminated throughout the substance of the gland. The deposit may be situated deeply, so that it is apt to point either into the perinæum or the rectum, or superficially, as regards the urethra, into which it tends to encroach. As is most usual in other regions, one here expects caseation and the formation of a tuberculous abscess. The *first signs of tuberculous disease* in the prostate may be frequency of *micturition* together with a *mucoïd discharge*. This frequency is made worse by exercise, and may precede the grosser diagnostic points by weeks or months. It is often the only sign which attracts the patient's notice to his condition, the discharge being discovered by the

doctor during examination. At this stage one must endeavour to exclude simple hyperæmia as the cause of the discharge, whilst a vesical or renal origin, total or partial, must be eliminated in connection with the urinary trouble. In the early stages of the disease it is very difficult, if not impossible, to achieve this end. More particularly so if there are no signs elsewhere of tubercle, past or present, to give a kindly hint. The disease progresses slowly but surely. The patient finds that he must empty his bladder at progressively shortening intervals. This symptom is more marked, and develops earlier when the focus is close to the urethral mucous membrane. But it is present, together with discharge, in a lesser degree even when the disease is situated more deeply. As in prostatic abscess and other conditions which cause obstruction to the outward flow of urine, we find that the *finale* to the act of micturition is incomplete—there is an after-dribble. This may be accompanied by pain, and, if much congestion or ulceration be present, by hæmorrhage. In the early stages of the disease every method of examination may fail to disclose either the site of the mischief or its nature. But sooner or later the thermometer will give a clue to the diagnosis which eye or hand had failed to grasp. As time goes by, the formation of an abscess or its offspring, an ulcer, bring conviction to the mind. But, as I remarked at starting, tubercle of the prostate usually forms but a part of a more extensive disease of the genito-urinary tract. The kidneys, the bladder, epididymis, or vesiculæ seminales sharing in the process. And it is the knowledge of this fact that tuberculous disease of the prostate, kidney, and bladder so often co-exist synchronously, and *have in common one cardinal symptom*, namely, *frequency of micturition*, that tends to make the diagnosis “of limitation” one of difficulty and doubt. Should the abscess discharge itself into the urethra, there will be a purulent discharge, often streaked with blood, whilst sloughs are voided with the urine. At this stage the amount of suffering varies, but is usually severe. Hæmorrhage may be profuse from the walls of the cavity. Urination is often agonising, and its frequent repetition, which has almost reached to the pitch of incontinence, is most distressing. In these cases which go on to a fatal termination, death may result from intercurrent tubercular mischief

in the lungs, kidneys, or meninges of the brain. Tubercle bacilli may be found in the discharge or urine.

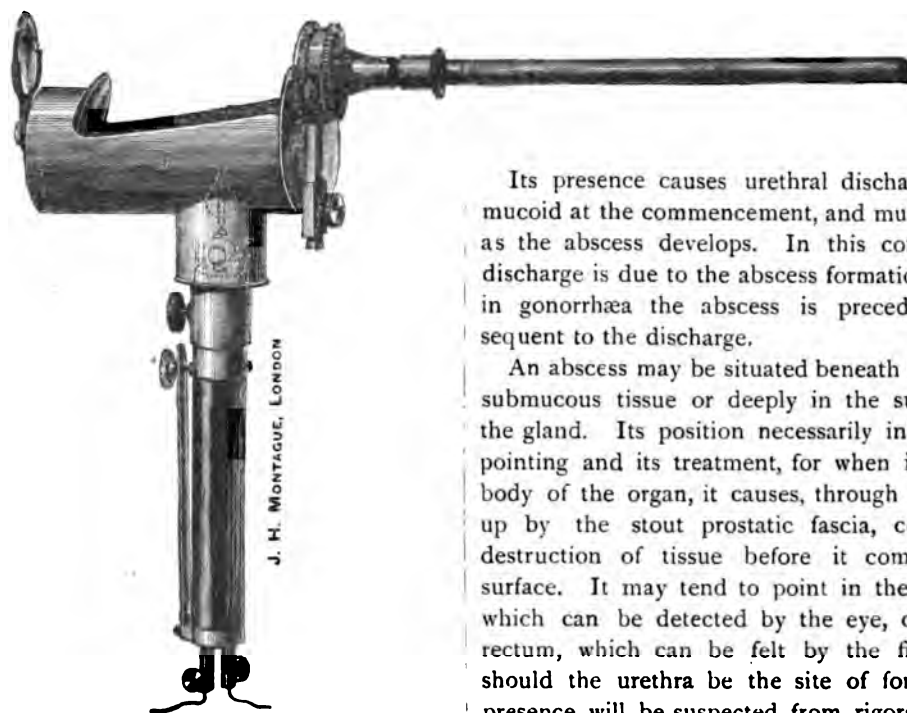
Treatment.—It stands to reason that in many instances the treatment can only be palliative. But in those fortunate cases where operative interference is permissible, the condition should be treated as it would be in other regions, namely, cut down upon and thoroughly scraped. If one is lucky enough to have a case in which it is possible to remove all the diseased tissue the wound will heal. But it is a slow process, often hindered by urine trickling through the track. In the more advanced cases, drainage of the bladder by a median cystotomy incision gives the greatest relief. In the earlier stages, when doubt obscures the diagnosis, one can but watch and wait. That tubercle in this region does become quiescent, as in other organs of the body, is, I am sure, a fact. I have seen a prostate that to digital examination was nodular, hard, and craggy, and which was but part of a tuberculous heritage, become dormant. To attain this end, rest is undoubtedly a most important factor, and should be of a physical and sexual nature. The latter is most difficult to secure, for the desire for procreation is strong in these subjects. The ordinary routine of building up the general health with tonics, together with a genial climate and a generous diet, are our slender resources in these early and doubtful cases. It requires great faith to believe that drugs have any specific influence on the course of the disease, but if I lean to one more than another it is to guaiacol carbonate, for I fancy it has done good in two cases. In another, where I had scraped the gland, and who had renal tubercle as well, I gave hypodermically 30 minims of the solution *serafon de gaiacol iodoformé* every other day for a fortnight. “Post hoc or propter hoc,” he improved. The prostatic wound healed, and the left kidney *seemed* to get quiescent. The operation was over four years ago and the patient is apparently well.

Polypi: (α) *mucoid*; (β) *fibroid*.—The diagnosis of a mucous polypus rests with its discovery. There are no distinctive diagnostic signs that I know which would point to its existence. The only two cases of mucous polyp that I have seen were discovered accidentally whilst examining the prostatic urethra to ascertain a cause for chronic muco-

purulent discharge. In one instance the discharge had lasted for over two years. On exploring by means of Leiter's endoscope a polyp was discovered. It bulged into the lumen of the tube. It was snared by means of an urethral écraseur and removed. The base was then touched with sulphate of copper. Nothing more was done. In fourteen days all discharge had ceased. The other case was a patient of the late Mr. Berkeley Hill. Here a discharge had existed for many months, and a similar condition to the foregoing was found

years ago. He was cured by operation. In this case there is no mention of a purulent urethral discharge, but as the sufferer had to resort to catheterisation he probably had a slight one. But it would be hard to say in such a case that the polyp rather than the instrument was the cause of the discharge.

Prostatic abscess.—This may apparently arise spontaneously in the course of parenchymatous prostatitis or may be induced by the passage of instruments.



LEITER'S URETHROSCOPE

in a like manner. The treatment employed, and the result obtained were identical with the first case. The necessary deduction is that the presence of the polypi was the cause of the discharge.

The very rare condition of fibrous polypus of the prostatic urethra may cause discharge. In a case related by Mr. Thomas Bryant the symptoms were profuse urethral hæmorrhage,* frequency of micturition, with marked obstruction and straining to void urine. There was progressive enlargement of the prostate. The patient attributed its origin to a gonorrhœa which he had contracted forty

Its presence causes urethral discharge, this is mucoid at the commencement, and muco-purulent as the abscess develops. In this condition the discharge is due to the abscess formation, whereas in gonorrhœa the abscess is preceded by and sequent to the discharge.

An abscess may be situated beneath the urethral submucous tissue or deeply in the substance of the gland. Its position necessarily influences its pointing and its treatment, for when it is in the body of the organ, it causes, through being pent up by the stout prostatic fascia, considerable destruction of tissue before it comes to the surface. It may tend to point in the perinæum, which can be detected by the eye, or into the rectum, which can be felt by the finger. But should the urethra be the site of formation, its presence will be suspected from rigors, a gradual increased frequency and difficulty of micturition which is very painful. Retention of urine may eventually occur. Free hæmorrhage at the end of urination is often met with. Some of the blood may flow backwards into the bladder and clot there. The effort of the viscus to expel these foreign bodies causes great suffering. If the clots are allowed to remain they may decompose and set up cystitis. When a prostatic abscess is present there is frequently turgidity of the penis, œdema of the prepuce, and fulness of the dorsal vein of the penis. Although a slight discharge is present in the early stage of a prostatic abscess, it is not uncommon for it to apparently cease later on, *puri passu* with the increase of the abscess.

* 'Med.-Chir. Trans.,' vol. lxxvi, p. 191.

The discharge probably regurgitates into the bladder, the exit *per urethra* being blocked by the swollen verumontanum. The general condition of the patient is that he wears an anxious look, his tongue is coated with a brown fur, and his temperature is high, whilst rigors point to a pyogenic condition.

Treatment.—The abscess may burst of its own accord into the rectum, perinæum, or urethra. But the latter event is usually brought about by efforts to relieve retention. They not unfrequently refill and burst only to refill again. Incision and drainage will affect the necessary cure, should they fail to heal naturally.

Prostatic calculi.—This rare condition may give rise to a mucoid or muco-purulent discharge. The stones may vary in number from one or two up to twenty-five or thirty or even more. They are most usually composed of phosphate of lime, but sometimes consist of carbonate of lime. They are born and bred in the prostatic ducts. They are of grey colour, smooth, hard, roughly triangular in appearance, and perchance faceted. Some may appear to be encysted. Their presence in the prostate causes fulness, bearing-down pain, frequency of micturition and discharge. They may set up retention of urine through causing swelling or from their size by impaction. Sometimes they can be felt by the finger *per rectum*. The introduction of a steel sound, if they are not imbedded in the substance of the prostate, will reveal their presence. They may be felt just as the beak of the instrument is passing into the bladder; or when the sound is in the bladder, if one presses the perinæum on to the instrument, they will be felt to grate against it.

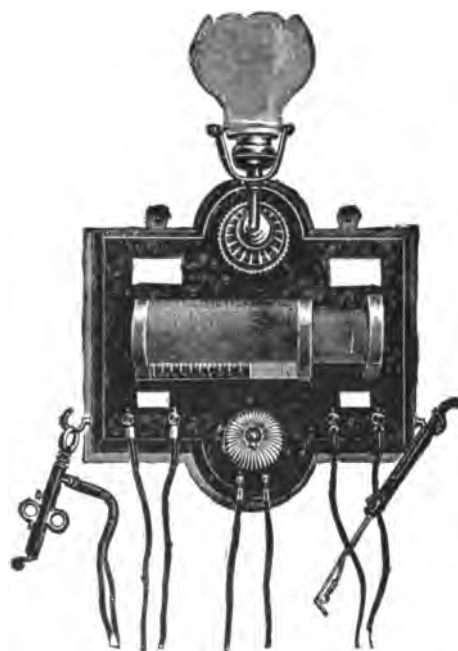
Treatment.—Unless the prostate should be honeycombed by the calculi, they may be removed by incising the gland in the middle line and scooping them out.

As the use of the urethroscope is entailed in the diagnosis and treatment of many of the foregoing conditions, a slight account of its structure and method of employment will form a suitable conclusion. There are more than one pattern of this instrument to pick from. The one perhaps that is best known and most generally in vogue is designed and manufactured by Leiter, of Vienna. For descriptive purposes the apparatus may be divided into two parts, though it is mechanically composed of more.

1. *The tubes.*—These are electro-plated cylinders, cut obliquely at the terminal orifice, and are roughly six inches in length. Their size varies from No. 26 French to No. 20 French. They are fitted with a removable ebonite plug to facilitate introduction. When the tube is in the requisite position the stylette is withdrawn.

2. *The light barrel.*—This consists of a metal barrel on a handle. The wires from the battery fix on to the latter at its free extremity, whilst the little electric lamp is connected to the end to which the barrel is adjusted. At the back of this light-bearing frame is a reflector, which is slightly movable for purpose of concentrating the rays, and when this portion of the instrument is fitted to the tube, for casting them down on to the surface of the urethra.

The ordinary domestic electric light, when present, may be utilized for illumination by means

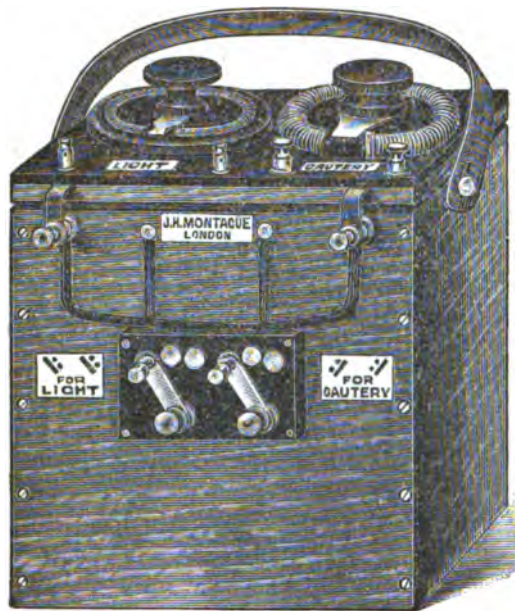


TRANSFORMER.

of a suitable transformer.* This contrivance diminishes the strength of the current to a degree suitable for these small lamps. These transformers are made either portable, to carry from house to house when visiting, or are fixtures in the consulting room. Light may be generated by means

* K. Schall, 55, Wigmore Street, London.

of batteries, such as the bichromate, Leclanché's dry cell, or from an accumulator. The first of these has the advantage of being easily kept in



ACCUMULATOR.

order, and it can also be used for cauterising purposes.

To view the prostatic urethra the dorsal supine position is insufficient. To effect this the patient must be posed as follows: he is seated, semi-recumbent against a back rest (an inverted chair and pillows make a good one) at the end of a surgical couch; this should either be *of*, or raised up *to*, such a height that his feet just rest on the floor. In this fashion the pelvis is oblique. The operator sits on a low stool in front of and between the patient's legs. Conveniently at hand he has his solutions and mops of wool on holders. It is advisable to have at least a dozen of these prepared.

The endoscopic tube is now introduced into the urethra, the largest size that it is possible to pass being chosen. The view gained by means of the lower numbers is very unsatisfactory. As a rule the tube goes with comparative ease as far as the triangular ligament, and then stops. It now becomes necessary to depress it, and at the same time exert a *gentle* pushing movement to make it enter the prostate. It is needless to say that no undue force must be used.

The next step before adjusting the barrel to the tube is to mop out the oil and perchance blood. The light-frame is then fitted on, and the urethral mucous membrane comes into view—provided that the light is properly reflected down to it.

Local applications can now be made should diseased areas be discovered.

For producing anæsthesia of the urethra a



GUYON'S SYRINGE.

Guyon's syringe is best. It hurts less than an Ultzmann's. If 30 minims of a 5 per cent. solution of cocaine be held in the pipe for five minutes the desired effect will be attained.

If solutions of cocaine have to be kept for any length of time it is advisable to add a little salicylic acid to them, otherwise they decompose.

Herpes Zoster.—Frich observed in a man æt. 75 a typical case of herpes zoster, affecting the entire right side of the neck and face, corresponding with the lower region of the cervical plexus. After ten days of suffering total paralysis of the right facial nerve suddenly set in. The pain and the paralysis gradually diminished, and finally disappeared after from two to three weeks' treatment by the galvanic current.—*Norsk Magazin for Lægeordenskab*, p. 1125, 1896.

NOTES.

The Use of Thyroid Extract and its Substitutes.—The thyroid gland having become a recognised and valuable therapeutic agent in the treatment of several grave, although comparatively rare affections, there have naturally been many studies made with the object of decreasing the bulk of the ordinary dose, and of preparing the gland or its extract so that its administration would be as unobjectionable as possible. With this in view there have been prepared capsules containing concentrated extract of thyroid, and tablets containing desiccated thyroid gland of such small bulk that one tablet containing two grains is equivalent to five grains of the fresh gland; and these tablets and capsules, or the desiccated gland itself, have been used for many months with the greatest possible satisfaction in the treatment of myxœdema, cretinism, and obesity of certain types.

Not satisfied, however, with the use of the gland in this form, certain German investigators have endeavoured to isolate from it its so-called active principles, and to market these derivative substances in small bulk, accompanied by the statement that they in every way represent the activity of the gland itself.

Clinical results, however, do not indicate that their theoretical views concerning the activity of these derivatives are correct; and not only is this true, but careful physiological study indicates positively that their influence upon general bodily metabolism is quite different from that exercised by the complete gland. It seems evident, therefore, that the entirely unobjectionable whole gland prepared in desiccated powder or capsule or in compressed tablet is the only means by which we ought to attempt to treat myxœdema and similar conditions in which this animal substance has been found useful.—*Therapeutic Gazette*, May, 1897.

Comparative Diagnosis in Pulmonary Tuberculosis by the Röntgen Rays.—Seventy-three cases are presented for the purpose of demonstrating that in the Röntgen rays and fluoroscope we possess accurate agents for diagnosing tuberculous changes of lung tissue in its various stages, using them not only as corroborative factors of results arrived at by auscultation and

percussion, but in some instances discovering isolated foci of infection not recognisable by ordinary methods.

In addition, the cases prove that the fluoroscope enables us to recognise more fully and accurately the degree, position, and relation of areas of infiltration and consolidation, and also delineates plainly the limits of these areas. It is unfortunate that as yet no satisfactory photographs have been taken of the images cast upon the fluoroscope plates.

In order that these observations might be of more value the examinations have been made by different physicians, and written notes thereof taken by different nurses, it having been my desire to obviate all danger of bias due to familiarity with the results of examination by the ordinary method before using the fluoroscope, and *vice versa*. In addition, after fluoroscope examinations have been made and notes taken in numerous cases, laymen have been requested to look into the fluoroscope and report the relative intensity of the transmitted light in different regions. Blue pencil marks were made by their direction, and they have invariably coincided with the professional examinations. The first thing that is noticed may be a comparative haziness or indistinctness of outline of the clavicle on the affected side.

In cases of slight infiltration of one or two apices there is a haziness or fog between the light and the observer, the clavicle in other instances appearing to have a gauzy veil thrown over it. When there is marked consolidation the transmitted light is relatively less, the edges of the clavicle are indistinct, or the bone may be invisible. When there is present the same pathological condition at both apices it is an easy matter, by comparing the two sides, at once to decide upon which the disease has made the most progress. Comparative shadows at the apices are generally seen more distinctly from behind than in front, by directing the patient to bring his shoulders forward so as to separate as widely as possible the scapulæ, and then placing the fluoroscope directly over the spinal column.

Ordinarily a practised eye can by these methods alone clearly distinguish areas of the most incipient infiltration; but if it is desired to be more accurate in defining their limits a metal rod may be placed evenly against the chest walls in front or behind,

and moved up and down with the fluoroscope until its outline becomes more distinct, which will indicate that the upper and the lower borders of the consolidation have been reached. If a pencil mark now be made along the edge of the rod, and subsequently percussion practised, the area of dulness will be found between the lines.

In cases of complete dulness, say, to the second interspace, with relatively less dulness for one or two interspaces below, a dark shadow will be seen over the first-named region, which will gradually shade off consecutively into haziness and normal reflex of light below, the area of haziness corresponding to the limits of relative dulness. In one or two instances slight haziness has been observed in spots which at the time showed no other physical signs of disease, but where they subsequently developed.

In cases in which the cavity is single, it appears as a bright reflex amidst an area of consolidation or shadow. This bright spot assumes the shape of the cavity. At times the observer can plainly discern a decidedly dark ring surrounding the dark spot.

In one case in which there were multiple cavities, the dense intervening tissue was shown in the form of dark streaks winding between the spots of bright reflex.

In another case the fluoroscope demonstrated a cavity which had presented no signs by percussion or auscultation, probably owing to obstruction at its outlet.

In other cases old pleuritic adhesions were seen as areas of absolute darkness, even more dense than the normal shadow over the cardiac region. Displacement of the heart downward and to the right was easily marked out. Cardiac dilatation was discovered in one case.

At different times we have outlined the convexity of the arch of the liver, which can be seen to rise and fall with expiration and inspiration. The cardiac pulsations are easily discernible.

A series of fifteen healthy cases was examined in order to familiarise the observer with normal reflexes and shadows.

A summary of the result of our investigations at the sanitarium shows—

1. Slight haziness indicates the beginning of tuberculous infiltration, and may or may not be accompanied by dulness.

2. Decided shadows indicate consolidation, the extent of which is in direct relation to the comparative density of the shadow thrown on the fluoroscope.

3. Circumscribed spots of bright reflex, surrounded by narrow dark shadow rings or located in the midst of an area of dense shadow, indicate cavities.

4. Intense darkness, especially at the lower portion of the lung, indicates old pleuritic thickenings over consolidated lung tissue.

A great deal depends upon the intensity and steadiness of the light, and for this reason a motor generator is better than a vibrator, as the latter gives a flickering light. An eight-inch-spark coil was used. Considerable practice is necessary before the eye can appreciate perfectly the finer differences of shades and outlines.

Medical Record, May 22nd, 1897.

Periodical Paroxysms of Hiccough.—S. Feilberg describes the case of a man æt. 42, who suffered for several months from periodical paroxysms of hiccough. The author finally found, at the base of the tongue, a tumour having three lobes, of which the central one, the largest, was about the size of a hazel-nut. These growths were due to a pronounced hypertrophy of the adenoid tissue at the base of the tongue, which caused, in addition to the hiccough, a continual sensation of fulness in the throat. There was also enlargement of the palatal tonsils, which were excised without producing any noticeable improvement. Ablation of the central lobe of the lingual tonsil caused considerable amelioration of the hiccough, which ceased entirely after the extirpation of the lateral lobes.—*Norsk Magazin for Lægevidenskab*, p. 1130, 1896.

Colouring Matter of Bile in the Urine.—M. Rasmussen recommends a new method to demonstrate the presence of the colouring matter of the bile in the urine. One cubic centimetre of urine is mixed with as much ether, and the mixture is well shaken with four to six minims of spirituous solution of iodine. The liquid then forms two strata; above is the ether with the iodine, and below the urine, which remains unchanged when no bile is present, but takes a brilliant green colour when biliverdin is present.—*Hospitals-Tidende*, p. 1200, 1896.

THE CLINICAL JOURNAL.

WEDNESDAY, JUNE 16, 1897.

CLINICAL LECTURE ON A CASE OF FRACTURE OF THE BASE OF THE SKULL.

Delivered at University College Hospital
By **CHRISTOPHER HEATH, F.R.C.S.**,
Holme Professor of Clinical Surgery.

GENTLEMEN,—I will commence my lecture by reading you a short account of this case of fracture of the skull from the house-surgeon's notes.

The patient was admitted at 3 a.m., having been knocked down by a cab, which was said to have hit him on the right shoulder and right side of the face. On admission he was unconscious, breathing stertorously, with respirations 30 to the minute. The pulse varied between 50 and 60. The conjunctival reflex was absent. The right pupil was moderately dilated, the left was contracted, and both were fixed. Blood was trickling from the right nostril. The skin was slightly grazed just below the right malar bone. Some urine had been passed, but no fæces. There was paralysis of the limbs and loss of knee-jerk. There was some discoloration of the skin just in front of the right thigh, but no other marks of violence were noticed. An ice-bag was applied to the head, and grs. v of calomel were placed on the back of the tongue. Hot-water bottles were applied to the feet, the temperature being under 95°. The breathing varied, sometimes becoming normal in character, and at other times stertorous. In the night the patient made some movements with his left arm.

About 6 a.m. bleeding increased, and blood flowed freely from both nostrils and threatened to clog the pharynx, which was cleared by repeated sponging. It was now noticed that there was proptosis on the right side, with swelling of both eyelids and cheeks. On each side there was a marked fluctuating swelling occupying the inner

half of the upper eyelid, of a purplish colour. The bladder was not distended.

At 8 a.m. the general condition of the patient was the same except that he was warmer, the temperature being 96.2°. There was proptosis on both sides, more marked on the right side, with increased swelling of the lids. The pupils were moderately dilated and fixed. The right nasolabial fold was less defined than the left. There was some drooping of the right angle of the mouth. The tongue was swollen and pushed over to the left. The bleeding was not so free. A Jacques catheter was passed easily, and 14 oz. of urine were drawn off. The urine was pale and acid, with a cloud of albumen. Knee-jerks were still absent. An enema simplex was given, but was returned without result.

From about 10.30 the patient's temperature gradually rose until 1 o'clock, to 99.2. The pulse became more rapid and feeble, 120, respirations became shallower, several times the bleeding increased and necessitated sponging of the pharynx.

At 2.30 p.m., or about twelve hours after the injury, the patient died from failure of respiration; the radial pulse could not be felt. Heart-beats continued for three minutes after cessation of respiration.

At the autopsy, on reflecting the scalp there was extravasation of blood beneath the temporal fascia on each side, but none under the scalp, and no fracture was seen. On removing the roof of the skull the dura mater was found to be unusually adherent; on the right side it was somewhat separated from the middle fossa, and there was extravasation of blood outside it of small size. On reflecting the dura mater, both temporo-sphenoidal lobes were covered by large masses of clot, the amount being larger on the right side. On the brain being removed, there was found a laceration of the dura in the right middle fossa close outside the foramen ovale, about three-quarters of an inch long, but it had not lacerated the meningeal artery. The dura was then stripped off and the fracture examined; it was found to extend from one side of

the skull to the other, through the middle fossa. Beginning on the left side in the temporal region, it followed the line of the great wing of the sphenoid to the sphenoidal fissure, then across the body of the sphenoid at the sella turcica, this part being comminuted, to the right foramen ovale. In the middle fossa the lines of fracture diverged, one extending upwards to the right temporal region, and sending a branch forward along the great wing of the sphenoid, as on the left side, to the sphenoidal fissure; the second branch extended across the roof of the tympanum almost as far as the lateral sinus, where it curved inwards to the jugular foramen. In the middle fossa the lines of fracture enclosed two large pieces of bone. The fracture of the petrous bone had lacerated the facial nerve, and opened up the internal ear. The membrana tympani, the malleus, and the incus were not damaged, but the stapes could not be found.

These notes form the history of a typical case of fracture of the base of the skull following a violent fall. The question now arises, how did that man fall, and how was the injury inflicted? In all probability the man fell on one side of the head, and I should think from the amount of injury it is probable that he fell on the right side, and that the bone was broken by the violent impact against the pavement, and then that the force of the blow spread across to the left side, involving both orbits. On the right side it extended backwards to the temporal bone, the facial nerve was lacerated, and though the tympanum was involved the membrana tympani was not ruptured.

Bleeding from the ear is one of the symptoms for which we always look in any case of supposed fracture of the skull; but in this case, though the symptom was absent, there was still an extensive fracture. The reason for its absence was that the membrana tympani did not happen to be torn: the blood did, no doubt, find its way out of the tympanum; but it found its way out by going down into the patient's throat through the Eustachian canal, and so was swallowed. There was certainly a considerable quantity of blood in his pharynx, and that blood no doubt came from the ear, as I have said, through the Eustachian tube. Both the orbits were open, and there was a large extravasation of blood on both sides. Immediately after the accident there was a little proptosis of the right

eye due to this extravasation, and about 8 o'clock there was also proptosis on the left side, and at 10 o'clock the eyelids on both sides were suffused with blood. It was clearly seen from the first that nothing could be done, and that the man would die in a few hours; and he actually died twelve hours after the receipt of the injury.

These cases of fracture of the base of the skull with hæmorrhage occurring at the time of the accident are almost invariably fatal, but there are a good many cases of fracture of the base where there is no hæmorrhage at the time, and it is those cases which may get well.

The injury was produced in this case by direct violence. When a man falls from a scaffolding and pitches down upon his head, the vertex coming first against the pavement, the whole weight of the body of a heavy man falling thirty or forty feet comes upon the basilar process of the occipital bone and breaks it through, and that is one way in which fracture of the skull occurs. Fracture also occurs similarly in children, in those cases in which a child falls out of a window; and these cases, as a rule, die of laceration of the brain with fracture of the base of the skull. But the more frequent way is that a man falling from a moderate height strikes his head at some point or other, sufficiently severely to fracture the bone, and then the fracture "runs round;" and here you see in this case that the fracture ran round, not only on the right side, but extended to the left, and opened up both orbits.

There is an old theory that a man can fracture the base of his skull by falling on his feet, but you must clearly understand that if a man falls from a height on to his feet, he breaks first his legs, and then his thighs, and that the pelvis and the vertebræ are fractured long before the base of the skull is affected, and, in fact, that it is impossible for the mere shock of falling on the feet to fracture the base of the skull. There have been very curious cases where the base of the skull has been fractured from patients falling violently on to the chin, sometimes not breaking the lower jaw, but driving the condyle of the lower jaw through the glenoid fossa into the skull and so fracturing the base, but they are very rare cases indeed. With regard to the different parts of the skull, of course the lines the fracture takes will depend entirely upon where the blow was struck. Sometimes the

fractures run right through the orbital plate. Though the orbital plate is very thin, the horizontal plate of the ethmoid is much thinner, being about as thick as an egg-shell, therefore it is not very surprising that fracture occurs from bodies pushed through the nose or from a blow on the front of the skull.

The symptoms the patient exhibits will vary much in different cases; in some they are simply those of concussion of the brain. The only thing, then, to say is that the patient has had a serious accident, he was insensible for a short time, and is now recovering, and we must be careful with him for a few days. I have seen such cases recover, although some days later I have been aware that there had been a fracture after all, and for this reason: the fracture may run through the temporal bone and involve in some way the aqueductus Fallopii, through which the facial nerve passes, and the proof of the fracture having occurred is that ten days or so afterwards the patient gets paralysis on that side of his face. The fissure, if it runs through the temporal bone, becomes repaired, and repair may occur with an excess of callus which causes compression of the facial nerve in the canal and produces paralysis. In another fortnight, when the callus is absorbed, the nerve resumes its function and the patient gets well. It is well to be on the safe side, and to prognosticate what may possibly happen, for if you have said that there is nothing the matter with the patient, and that he is able to go about his work, on anything happening, such as this facial paralysis, there is much surprise expressed at such an event supervening. But if you have warned the friends and the patient, as you ought to do, that certain symptoms may develop and that the patient should not go to work, on anything untoward occurring you are on the safe side.

In the case we are considering to-day there was a fracture of the base of the skull and extensive hæmorrhage. Where did that hæmorrhage come from? Bleeding may be from the middle meningeal artery, but the effusion in that case is outside the meninges, and the blood accumulates and forms a clot producing well-marked symptoms of paralysis. But in this case the meningeal artery was not torn, and the hæmorrhage came from the brain, for there was no doubt that there was some laceration of the brain substance here, which

would naturally occur in such a violent injury. The brain is very vascular, and it is easily to be understood how the blood in this case has permeated all the surrounding tissues. You can understand how the blood first formed a clot, produced those signs of paralysis of which we have heard, and lastly it would find its way into the orbits, and produce the proptosis which was so characteristic in this case. In such a case you cannot get at the source of the hæmorrhage, you cannot trephine; you can only let the patient be, and very soon the end comes.

In the slighter cases, where there is a fracture of the base of the skull, and some irritation from the laceration of the meninges, you are apt to have the occurrence of meningitis. We do not happen to have had a case of that kind lately in the wards, but it is well always to bear in mind that these cases of head injury are apt to take on meningitis within forty-eight hours after the injury, and you ought to anticipate it. The way to anticipate this meningitis is to clear the bowels out, and in the second place to put an ice-bag on the head to ensure quiet, and insist upon absolute starvation diet. Mistakes are often made in these cases by not looking carefully after the nurses. Nurses with the best intentions sometimes force nourishment on such patients, but judicious starvation of a brain case is really one of the most important elements in the treatment. By starvation diet I mean milk and water and nothing else for the first forty-eight hours or so, and then perhaps a little bread and butter, and then very gradually and very slowly to increase the diet. You must be guided by the temperature and the general symptoms before you let the patient take anything really solid in the way of nutriment.

With a patient who is getting over the first shock, and with meningitis developing, you will find that his temperature goes up. At first, when entering the hospital, the temperature of these patients is below normal, they are suffering from shock; after that the reaction causes the temperature to go up, and in these cases purgation and ice-bags are good treatment. But when you find the patient with a temperature going up to 103° and 104° , with a good deal of headache, a quick pulse, and a great deal of heat on feeling the head, you may be sure that he is developing meningitis. The patient will probably die if you are not prompt in your treat-

ment, and you will find at the post-mortem the surface of the brain smeared with pus which the meningitis has caused. I do not say that in every case of threatened meningitis you will be able to prevent it, but the tendency is nowadays, I think, not to be active enough in the treatment. You should clear the bowels out and use an ice-bag, and if the temperature keeps up I should administer calomel. Calomel was formerly given very freely, but the present generation of surgeons seem to have lost their faith in it, and yet if you have a case of head injury where the temperature is keeping up and the patient is in a highly feverish condition, and probably developing meningitis, by administering calomel until the gums are affected you may possibly save your patient's life. I advise you to give 2 grs. of calomel every four hours without any opium. If you give opium in these cases you complicate them slightly, and you are not sure how much of the symptoms is due to the opium and how much to the coma following the injury. In this treatment by calomel, when diarrhoea has developed, you must reduce the dose of calomel to every six hours, and then watch for the gums becoming affected. The patient must be given calomel to just such an extent that while affecting the gums you do not actually salivate the patient, but he must be brought under the influence of the mercury. I have great faith in mercurial inunction in cases of syphilis, and in cases of head injury mercurial ointment rubbed into the thighs and loins will do good.

I have seen patients who have got well under that treatment. The answer to that of course is, Would not the patient have got well without any calomel? I can only say that possibly he might; but when you find a large number of cases not treated by calomel dying of meningitis, and that every now and then a patient who does take mercury recovers, I think it reasonable to draw the conclusion that the mercury had something to do with the cure. I would therefore recommend you, in all appropriate cases where a high temperature exists with a full hard pulse, to give mercury; and I would not hesitate to bleed in a suitable case by leeches or from the arm.

As to the flow of blood from the ear when the patient is first admitted, the cases in which this occurs are by far the most common; there is more or less severe concussion of the brain, and then

the bleeding is noticed from the ear. If this bleeding occurs in some quantity it is a matter of great consequence, but the mere passage of a little blood from the ear may be nothing, a mere laceration of the lining membrane of the ear may cause it. If, however, the blood marks the pillow, and if there is a discharge of a watery fluid from the ear as well, there can be no doubt that it is a case of fracture of the base of the skull with escape of the cerebro spinal fluid. There have been many disputes concerning this cerebro-spinal fluid; it was said to be simply the serum of the blood, then some said it was the liquor Cotunnii from the labyrinth of the ear, but now it has been proved that the fluid is the cerebro-spinal fluid, and the explanation of how it gets out is very simple. If you examine the skull you will find the internal auditory meatus with a process of dura mater containing in it the auditory and facial nerves, and a fold of the arachnoid going round the nerves. That process is prolonged upon the nerve into the foramen to the point to where the auditory and facial nerves separate. The fracture in the temporal bone passes through the petrous portion of the bone across the tympanum, and tears through that little process of arachnoid upon the nerve. The fluid therefore gravitates from the subarachnoid space into the tympanum, and finds its way out of that cavity, and in this particular case before us to-day it was down the Eustachian tube; but as the membrana tympani is generally torn through, it usually comes out of the ear. The pillow is noticed to be wet with the watery fluid, which is recognised at once as the cerebro-spinal fluid. Up till quite recently we were content to let that cerebro-spinal fluid run away, perhaps examining it sometimes to see that it really was cerebro-spinal fluid, but nothing was done to these cases and patients were allowed to continue to die from septic meningitis. However, at last it was suggested by some one that if the external auditory meatus were cleansed, and if it were plugged with an antiseptic dressing to prevent septic material being introduced into the meninges, it would probably be a great advantage. Accordingly, now-a-days, cotton wool is dipped into carbolic acid lotion and wrapped round a probe, and with it we wipe out the external auditory meatus. I advise you to do it

with a probe and not with a syringe, because by syringing you might drive some of the deleterious material into the cavity of the cranium. Having carefully mopped out and cleaned up the meatus in that way, you blow a little iodoform into the cavity, and then put a small piece of iodoform gauze or wool into the auditory meatus. After a few hours that gets soaked with the fluid, and then it is renewed. There is no question that since this practice came into vogue, some ten years ago, the mortality of these cases of fracture of the middle fossa of the skull has been greatly reduced by this means.

In all these cases, if things go well, the fluid runs through the fracture for some days and then stops, and the patient gets gradually quite well; but throughout the case there is always a danger of meningitis, and after he is well you must be extremely careful to guard against exposure to the sun. These patients are very apt, having been kept on very low diet for a long time, to take some alcoholic liquor directly they can, and if there is one thing that upsets these head cases more than another it is the taking of liquor of any kind whatsoever. Merely a pint of beer will give these men a bad headache, make them sick, and there may be further mischief. You will find that the tendency for liquor to "fly to the head" remains for a very long time, and that often these men ever afterwards cannot take the amount of liquor that they took before the accident.

CLINICAL NOTES ON A SIMPLE METHOD OF OPERATING ON VARICOSE VEINS OF THE LEG.

By ARTHUR E. BARKER, F.R.C.S.,

Professor of the Principles and Practice of Surgery at
University College, and Surgeon to University
College Hospital.

HAVING for some time past adopted a method of operating on varicose veins of the leg, which has given me excellent results, although departing in a measure from the usual routine, it appears desirable to make a note of it in this Journal, in order that

others may test it. Its chief merit consists in the fact that no foreign body is left in the wound in the shape of ligatures or sutures, and that it saves much time. The latter point is one of considerable importance, not only to the patient, who is thus saved any prolonged anæsthesia, but to the surgeon who now-a-days is called on to do a very great many of these operations, both in hospital and private.

It goes without saying that the method is only proper where perfect asepsis can be secured. The leg is first washed, shaved, and carbolicised. Then the saphena vein is exposed as high as possible in the thigh, preferably at the saphenic opening, as recommended by Trendelenburg, by an incision of about two inches long. The vein is then caught above and below by two ordinary serrated Wells forceps, and a piece cut out between them. A sponge is then placed in the wound, and the vein is similarly treated lower down in three or four places, *i.e.* wherever markedly varicose. When all the wounds have been dried, and all bleeding stopped with catch forceps, the latter are removed one by one, as an ordinary white gauze bandage, which has been steeping in 1 to 20 carbolic lotion for forty-eight hours, is applied to the limb in the usual way well below the lowest wound, and is wound round until it is about to cross the lowest wound. The edges of the latter are held together with the left thumb and first finger, and the bandage is carried across them. When the fingers are removed the edges will be seen through the transparent bandage to be coapted very accurately. If this is not the case at one spot or another, a probe slipped under the bandage through which the edges can be still well seen will easily adjust the edges, while the gauze is held taut. Then the bandage is carried up the limb, each wound being similarly treated. Over the gauze I usually lay a strip of salicylic wool, and retain it with firm turns of an ordinary bandage to keep up steady elastic pressure. This dressing is left on until the tenth day, when the wounds will be found to be closed by a linear scar as accurately as if they had been stitched, and only require a little wool and collodion for a few days longer.

The advantages of this method, which is only an application of that which Dr. Credé of Dresden applies to amputation wounds, are its simplicity, its rapidity, and that the patient has no removal of

stitches to look forward to. There is also the advantage that no ligature is left in the wound. Such ligatures occasionally, though rarely, give rise to pain for weeks.

The only point to be specially attended to is the arrest of hæmorrhage from collateral vein branches as well as from the main trunk, and even pressure.

Those who become familiar with this method will, I feel confident, never go back to the more elaborate ligaturing and suturing.

From the patients' point of view, too, there is a great gain, and when told the day after operation that there is nothing further to be done to the wound except to remove the dressings, they are more than satisfied.

In some cases, where patients are inclined to vomit after the anæsthetic, a fine ligature may be applied to the upper end of the saphena vein, as the forcing of sickness may break the adhesion produced by the forceps. But such cases are rare, and firm bandaging meets them.

DEMONSTRATION OF CASES

At the Monthly Meeting of the North-West London Clinical Society, held at the North-West London Hospital on April 28th, 1897;

Mr. JACKSON CLARKE in the Chair.

Pseudo-Hypertrophic Paralysis.

DR. GUTHRIE showed a boy æt. 9 years, the subject of pseudo-hypertrophic paralysis. The patient was the third of six children, five of whom survived. No other members of the family had any such condition. The boy had never been able to walk very quickly, or to run, but nothing was considered to be particularly the matter with him until four months ago, when he was noticed to fall about frequently, and seemed weaker on his legs than formerly. The lad was fairly intelligent, and could not be said to be imbecile in any way. The facial muscles were not affected. Of the muscles of the neck, the trapezius and the sterno-mastoids were normal. On the other hand, there was distinct firmness and enlargement of the infraspinatus muscles on both sides. There was also, in his

opinion, some deficiency of the serratus magnus, causing the scapula to project, but not to a marked degree. He could not make out any deficiency in the latissimus dorsi. The deltoids and the lower half of the pectoralis major were small. As the patient stood, it would be noticed that there was a certain amount of lordosis. The buttocks were somewhat firm, but not markedly so. There was some enlargement of the vastus internus, but the thigh muscles generally were not so well developed as they should be in a child of his age. The calf muscles were distinctly more prominent and firm than normal. His knee-jerks were entirely absent. He walked with his legs rather far apart, and raised the heels abruptly during locomotion. It would be seen that his manner of up-rising was very typical of the disease: he first got on to his hands and knees, then raised himself by placing his hands on his knees, so as to relieve the muscles of the back from supporting the weight of the body. Pseudo-hypertrophic paralysis was one of the muscular dystrophies, and belonged to the same category as idiopathic muscular atrophy, the only distinction being that in the former cases there was apparent enlargement of the muscles owing to infiltration of fat and connective tissue between the strands of muscular fibres. In the atrophic cases there was no such infiltration of fat, but otherwise the muscular conditions were precisely the same. Later on the pseudo-hypertrophic varieties led to the atrophic varieties.

As to the etiology, one supposed it to depend on some embryonic deficiency or taint. He had mentioned that none of the members of this family had suffered as the present patient; but all the children were young, and it was therefore possible that later on some of them might be affected in the same way. It happened sometimes that the disease was not recognised until towards adult life. Dr. Gowers mentioned a case of a girl who was considered well until the age of twenty, when she became weak on her legs. But it was remembered afterwards that as a small child at school she was chaffed and teased on account of her "tea-kettle" calves. Of course the prognosis was fairly bad, because the disease was slowly progressive, and after a time the muscles became atrophied. The weakness became greater, and those muscles which were not antagonised would produce deformities; the child would be-

come bedridden, and would probably die young of some intercurrent affection.

The only treatment which seemed likely to produce good was massage and appropriate exercises. By these means one might perhaps stimulate such muscular fibres as remained, and keep the disease at a standstill for a time.

Peripheral Neuritis.

Dr. SWAINSON showed a man just past middle life, who had been a very heavy drinker, and last August had pains in his left foot, followed a few months later by similar pains in his right foot. He was laid up for a month before coming to the hospital, which was three weeks ago. On admission it was found that the extensor muscles of the left leg were paralysed, that there was a considerable burning pain over the dorsum of the left foot, and some pain in the right foot. There was also weakness of the extensor muscles of the right leg. The peculiarity of the case was that although it was alcoholic, the left leg was far more affected than the right. The knee-jerks were present, and appeared to be exaggerated. The plantar cremasteric and epigastric reflexes were present. The left foot dropped. All the muscles acted to the interrupted current, *i.e.* faradic. The patient had had numbness in his left hand, and a cutaneous eruption in his right leg. There was pigmentation. There was no bladder trouble.

In reply to Dr. Cagney, Dr. SWAINSON said there had been no pain in the anterior tibial muscles, but there had been anæsthesia and pain over the dorsum of the foot. The anæsthesia on the left side had extended up the leg nearly as high as the knee, principally on the outer side. There was no anæsthesia on the right leg.

Dr. CAGNEY said that personally he attached more importance to the loss of the knee-jerk in alcoholic neuritis than in any other disease except locomotor ataxy. He remembered one case in which a history of drink was entirely denied. The subject was a young gentleman æt. 22, who was brought to him by a friend, who said he had lived and dined with him continually, and that the young man had practically never been out of his sight. The patient was a most energetic business man, and sought advice because his liver was said to be out of order, which was the fact, but in the course of examination it transpired that he had

had some tingling sensations in his feet. This led him (Dr. Cagney) to find marked tenderness, especially in the anterior tibial muscles, and a condition of anæsthesia which was very suggestive. There was some paresis of the dorsal flexors of one foot. Taking into consideration the age of the patient and the circumstances, he felt sure it could only possibly be a case of alcoholic neuritis. He therefore told the young man that he believed he had been drinking heavily. But it was denied, and his friend said it was impossible, as he would otherwise have witnessed the excess. The patient was thereupon ordered to bed, and told to drink only milk and soda. The knee-jerks were at this time present, and even exaggerated. Within a week the knee-jerks had disappeared, and the patient was completely paraplegic. He went through a severe course of pseudo-alcoholic tabes, and for two or three months was absolutely unable to move. He now freely admitted his heavy drinking, and his friend chanced to remember that he had been away from him for a period of three months. He mentioned the instance as evidence of the strength of his own conviction, for he had predicted the loss of the knee-jerk, and submitted the event as a test of the accuracy of his judgment in the difficult circumstances detailed. In men of the character of the patient before them he thought alternatives occasionally presented themselves. He believed that chronic forms of anterior polio-myelitis were common enough, were often not extensive in degree, and were usually accompanied by changes in the peripheral nerves. He had seen a case in a man who had been attacked when laying flags in the streets. He had acute polio-myelitis, from which he recovered, but the incidence of the paralysis was entirely upon the muscles, which caused the foot-drop; there was no anæsthesia in that case. The fact of there being anæsthesia on one side was an important matter in the present instance.

Dr. GUTHRIE agreed with Dr. Cagney about the extreme rarity of the cases in which the knee-jerk was absent in alcoholic neuritis, but because the knee-jerk was absent he did not agree that the case should not be regarded as alcoholic. In infantile paralysis there was often paralysis of the extensor muscles of the foot, but if the extensors of the thigh were not involved, the knee-jerks might still be retained. Sherrington had shown that one

might divide all the nerves which supplied the quadriceps femoris, and the knee-jerk would be retained until the nerve supplying the vastus internus was divided. Judging by the symptoms of the present case he was inclined to regard it as one of alcoholic peripheral neuritis, rather than one of central mischief.

Peripheral Neuritis.

Dr. CAGNEY showed a woman æt. 26, by occupation a nurse, the subject of peripheral neuritis, for which he would not attempt to assign a cause. In November last she had intolerable pain, and her hand was brawny and swollen to twice its ordinary dimensions, and the skin at the backs of the fingers was glossy. The brawniness and œdema extended above the elbow, so that the whole of the forearm was involved; and though cold to the touch, it was at the same time the seat of burning sensations. It has been suggested that it was an interesting case of vaso-motor paralysis, but this he thought was a physiological rather than a clinical conception. He (Dr. Cagney) found that the illness dated from a period of seven years before, and at first affected only the skin and subcutaneous tissues. At that time the patient had chilblains on the left hand and forearm—an unusual place for chilblains,—but the expression sufficiently described the condition. Sensation was apparently normal in the present attack, but the thickened skin would not transmit impressions readily, and there was a degree of dulness due to this. There was wasting of the left deltoid and the teres minor, but not to any marked extent; there was also some wasting of the extensor muscles in the forearm. On deep pressure between the acromion and clavicle the patient complained at one time of pain, though she did not do so now. There was marked tenderness on pressure over the quadrilateral space and over the whole extent of the musculo-spiral nerve. Extension of the elbow and wrist were noticeably impaired, and the ends of the humerus and ulna at the elbow were painful and somewhat enlarged. There was a tender point about the middle of the belly of the supinator longus. At that time there was a decided reaction of degeneration in the front and back part of the deltoid. There was marked inability to extend the wrist, and a difficulty in pronation also. She was admitted into hospital some time later, and had been

an in-patient since. In his daily watching he had had opportunities of seeing interesting but minute changes. The result was, he was able to substitute clinical conceptions for those which were based on experiments in the frog, &c. Mr. Swainson mentioned in connection with his case that the galvanic current had not been used to diagnose it. Now it was an error to affirm that there is a reaction of degeneration based on the test with the faradic current alone. Absence of contraction to the faradic current meant nothing more than a destruction at that time of the function of one of the muscular nerves. This was a different thing from the reaction of degeneration. It was only by the qualitative changes in muscle that one could predicate the degenerative reaction. Here it was present in minute degree, but unmistakably. As to treatment, he had swathed the arm from the finger-tips to the shoulder in pinewood wool. He did not claim, as the enterprising manufacturer did, that there was any electric property about this substance, but it kept the limb warm very effectually, and it had been used on this patient during the past winter with very good results. From the very first slight manipulation—so-called massage—was employed, and a powerful galvanic current passed once or twice a day, with large injections of strychnine into the muscles at different parts of the affected limb. After that treatment he was glad to say the patient was recovering fast. There remained a contracture at the elbow.

Dr. GUTHRIE thought the case an extremely interesting one. The affection of the shoulder muscles in the way mentioned implied the condition known as Erb's paralysis, which was due to injury to the brachial plexus, especially that part which supplied the muscles of the shoulder. He would therefore like to know whether Dr. Cagney could exclude all possibility of injury to the brachial plexus.

Mr. JACKSON CLARKE said he had noticed in injury to the median nerve or fracture of the humerus that there was a similar condition, a stage of hyperæsthesia with reddening of the skin, accompanied by muscular paralysis, then a stage of lowered sensibility in the limb, accompanied by muscular contracture. They had been paralysed before they contracted. That offered so marked a contrast to what one saw in anterior polio-myelitis, where the muscles which were paralysed were

over-stretched by their opponents, that he had wondered whether one could make two classes of cases,—one of peripheral neuritis, localised in large nerves with contraction of the paralysed muscles following, and another class where stretching of the paralysed muscles occurred.

Dr. CAGNEY in reply said, if pressure by the scalenus muscle on those nerves of the fifth and sixth cervical roots was sufficient to produce Erb's paralysis by itself without any predisposition, Erb's paralysis would be far more common than it was. As to the progress made by the patient, the atrophic condition had completely passed away, and she had no more chilblains, from which she previously suffered. There was now no pain whatever in the skin. The only pain she had was on passive extension of the contracture of the biceps and supinator longus. He had called the attention of the profession to a very rare instance of a combination of the two kinds of cases mentioned by Mr. Clarke. An old lady suffered from cerebral hæmorrhage eighteen months before, leading to complete hemiplegia. She had been at that time under the late Professor Charcot. Every day it was asked why was there no contracture. He (Dr. Cagney) came to the conclusion that the explanation of this unusual phenomenon was in the fact that she was also the subject of diabetes. On applying the electrical tests he found she had peripheral neuritis in the limbs, which ought to have been contracted. He proceeded to show how contracture might be incompatible with peripheral neuritis.

Vascular Lesions in Pons.

Dr. CAGNEY showed a man æt. 64, who all his life had suffered from arthritism. The case was sent to the Hospital for Epilepsy and Paralysis as a case of general paralysis, which it certainly was not. The joints had been deformed, and he pointed out that the patient had Dupuytren's contraction. The illness began three months before he came under his (Dr. Cagney's) care on the 17th March. It commenced suddenly with three seizures. With the first attack, which was slight, he lost his speech, and recovered from that. The next and following seizures occurred closely on one another, and he could not ascertain what was the precise period which intervened. The third attack was followed by complete aphasia, from

which the patient partially recovered. At present his speech was characteristic. There was facial asymmetry, due to atrophy of the right side of the face. The angle of the mouth on the right side dropped, and the naso-labial furrow was less marked on the right than on the left. The pupil was markedly contracted, but was more so once. The tongue protruded to the left side. There was no marked loss of sensation, and no paralysis of any of his extremities. Laryngoscopic examination showed that when admitted there was abductor paralysis of the right vocal cord, and there was now impaired movement of that cord. His knee-jerks were normal, and when he laughed they would notice want of control over the glottis. The diagnosis in his opinion was vascular change, certainly not hæmorrhage, in the lower part of the pons and medulla oblongata. The affection of speech was no doubt due to subcortical injury.

DEMONSTRATION OF CASES

At the West End Hospital for Diseases of the Nervous System.

By HARRY CAMPBELL, M.D., F.R.C.P.

CASE 1.—This patient is 65 years old, and is suffering from paralysis agitans. The body, you will observe, is bowed forwards, the arms are flexed, the wrists resting upon the waist. You will notice that the expression of the face remains the same throughout: it does not alter from moment to moment. When he turns round, the head and body move as if they constituted one rigid piece. This is a characteristic feature, and is due to rigidity of the muscles.

In a typical case of paralysis agitans, the patient gets up very slowly, and then starts off walking, first at a slow pace, and then quicker and quicker. This peculiar gait is called festination, and was attributed by Trousseau to the patient running, as it were, after his own centre of gravity, owing to the bowing forward of the body; but this cannot be the true explanation, because festination is often even better displayed when the patient walks backwards, and that when there is considerable

forward bowing of the body. You see this patient has not the slightest tendency to festination.

Now notice the tremor. The hand is in a position very common in this disease, that of interosseal spasm, the fingers being flexed at the metatarso-phalangeal joints, and extended at the other joints. There is also flexion at the wrist- and elbow-joints. The rhythmic movements are due to rhythmic spasm of the interossei and to rhythmic flexion of the wrist- and elbow-joints. There is also rhythmic movement of the lower jaw—a somewhat unusual feature in this disease. You notice that when the patient attempts to pick up this piece of paper the spasm ceases in the member employed, the spasm in this respect differing from that of disseminated sclerosis, which is increased by voluntary effort.

Briefly, the characteristic features of paralysis agitans are these. You have bowing forward of the body, the arms held at the waist, rhythmic tremor diminished by voluntary effort, stolid facial expression, deliberate utterance, festination, and movement of the body as if all the joints were welded together. The disease, therefore, manifests itself in many ways, and I would particularly impress upon you that the rhythmical movements constitute only one among many symptoms of the disease, and are not, indeed, an essential feature: a person may have the disease without any tremor.

It is frequently said that paralysis agitans is brought on by fright. I doubt if the disease itself is ever thus induced; but that fright may bring out the tremor in an individual who is already afflicted with the disorder is certain. Thus Charcot describes the case of a woman in whom the agitating movement was brought on by seeing her husband's horse return riderless to the barracks: and Gowers that of a woman in whom it was induced by a jet of cold water unexpectedly striking against the hand. A common symptom of paralysis agitans, which our patient does not display, is an inordinate sensation of heat, so that even in the depth of winter a fire cannot be borne, and the patient will sleep without a blanket.

One word about the seat of the lesion in this disease. It is probably in the cortex. First, because the movement ceases during sleep, when the activity of the highest centre is more depressed than that of the lower ones; and secondly,

because hemiplegia has been known to remove the movement on the affected side.

CASE 2.—This case I will just describe to you, and I leave you to diagnose it for yourselves. I think you will agree with me that it is a very interesting one. This patient had syphilis fourteen years ago, and he came to the hospital last Friday complaining of numbness and pains in his hands and fingers. One's first impression was that it was an ordinary case of dead fingers, but it soon became evident that it was something more serious than this. (The patient was asked to pick up a piece of paper from the table with either hand, to unbutton and button his coat, to close his eyes and touch the tip of his nose with the index finger of each hand.) I think you will all agree the patient has decided inco-ordination in the upper extremities. (To the patient.) Tell me if you feel that. ("No.") There is, you see, a certain amount of anæsthesia to touch sensations in both hands, and it is possible that careful exploration of the skin would discover other areas of anæsthesia. (Touching the patient's palm with a needle.) Do you feel that? What does it feel like? ("A pricking.") Just call out when you feel it. (Patient called out after an interval of some seconds.) You see there is delayed sensation to pain. Cases of locomotor ataxy have been recorded in which the delay has lasted as long as eighteen seconds. (To the patient.) Now close your eyes, put your toes and heels closely together. Now turn round.—You are probably surprised to find that there is not the slightest inco-ordination of the lower extremities. (The patient was then seated on the table and his knees were tapped.) There is, you will observe, no knee-jerk. (To the patient.) Have you ever had shooting pains in your legs? ("Once or twice.")

One of the first things that struck one in looking at this patient was the smallness of the pupils. I may tell you that they do not react to light, but they do to accommodation, and that he has occasional diplopia. Then he tells me that his sexual power is quite gone, and he has a sense of tightness round the waist.

I take it that you have all made the diagnosis of locomotor ataxy. This is a very rare case—one in which the inco-ordination, lightning pains, and anæsthesia have developed in the upper extremities before the lower.

CASE 3.—This child has congenital spastic paraplegia. You notice the cross-legged progression; it is due to spasm of the adductors of the thighs, which causes the advancing leg to be moved across the other one. The child, you will notice, stands on its toes, owing to chronic spasm and contraction of the calf muscles; both feet are, in fact, in a position of equinus; it is impossible to flex the foot on the leg. You notice that the knee-jerks are very considerably exaggerated. It is impossible to get any ankle-clonus in this case; but Mr. Cotterell, you may remember, showed a similar case here a short time ago in which this phenomenon was very marked. You see that the elbow-jerk and wrist-jerk can be obtained on both sides. There is some weakness in the upper extremities. The child cannot even crawl. She has perfect control over the sphincters.

CASE 4.—This little girl is $4\frac{1}{2}$ years of age, and is suffering from the same disease as the last. The adductor spasm is particularly well shown. The child has also exaggeration of the knee-jerk, but no elbow- or wrist-jerk can be elicited; nor is there, as there is in the last case, any weakness of the upper extremities. Both these children, the nurse tells me, complain of cold feet.

These two cases, I think, are fairly typical examples of congenital spastic paraplegia, of which the most characteristic symptoms are rigidity of the muscles of the lower extremities, especially of the adductor muscles of the thigh, and of the calf muscles (causing crossing of the legs and talipes equinus), and exaggerated knee-jerk.

With regard to the origin of these cases, it would seem that some of them are due to an injury of the motor centres during birth. You may remember that in a case that Mr. Cotterell trephined and showed here there was evidence of an old meningeal hæmorrhage over the motor area. Other cases have a different origin, and are not yet understood. I will merely observe in this connection that the pyramidal tracts whose faulty structure is responsible for this disease are late in development, their fibres being devoid of medullary sheath at birth.

CASE 5.—The next case is one of primary lateral sclerosis in an adult. The patient is a woman 52 years of age. She is married, and has had fifteen children. Eight months ago she noticed a cold sensation, and weakness in the right arm.

The right leg was next affected in the same way, and then the left arm and leg. She has been unable to walk for the last six months. During the last six weeks she has had rheumatic pains in her knees, hips, shoulders, and elbows.

Let us now proceed to the examination of the patient. She cannot stand. The legs are stiffened in extension, so that if she could walk, she would wear her boots out at the tips. It is only with great difficulty that one is able to flex the legs at the knees. The knee-jerks, you observe, are exaggerated, and ankle-clonus is well developed. Sometimes one is unable to elicit it. Plantar reflex is present. There is no affection of sensation in the lower extremities, and there are no bladder or rectal troubles. Evidently the lesion in the lower part of the cord is confined to the lateral columns. Now let us examine the upper extremities. The right one is almost completely paralysed; the left one has more power. One gets no evidence of rigidity at the elbow- and shoulder-joints, but the fingers are somewhat rigidly fixed in such a position as to cause the hands to be "claw-shaped." Elbow-jerk and wrist-jerk are pronounced. Examining the hands more closely, we get evidence of atrophy of several of the smaller muscles, notably of the interossei, and it is chiefly to paralysis of them that the claw-shaped appearance is due, this condition being further favoured by the rigidity of the long flexors. On faradising these small muscles it is found that their response is very sluggish. Sensation in the upper extremities is normal. Hence we conclude that there is lateral sclerosis in the cervical region, together with some involvement of the anterior horns causing muscle atrophy. Examining now the face, slight weakness of the right side is made out, and on percussing the right masseter while the mouth is open, the lower jaw, you observe, is suddenly jerked upwards. There is, in fact, increased myotatic irritability on this side. It would therefore appear that there is some sclerosis of the "pyramidal" fibres belonging to the cranial motor nerves. The case belongs to the class described by Charcot as amyotrophic lateral sclerosis. If the disease runs the classical course as defined by him it will terminate in bulbar paralysis.

CASE 6.—The boy that is coming in now is $2\frac{1}{2}$ years old, and he has hemiplegia. You will notice the right hand is pronated and clenched,

the thumb being strongly adducted. The right hand is nearly always in this position. The nurse tells me that the spasm relaxes during sleep, and that it is always exaggerated when he tries to seize anything. You notice that he walks on the toes of the right foot, the rigidity here causing extension instead of flexion; also that there is exaggerated knee-jerk, and distinct wrist-jerk and elbow-jerk on the affected side. There is no obvious facial paralysis. Hæmiplegic facial paralysis clears up very rapidly in children.

Here, then, we have a case of infantile hemiplegia, with rigidity of the paralysed members and increased myotatic irritability, showing the supervention of secondary lateral sclerosis. This child will probably improve considerably.

CASE 7.—This is a very similar case, but the paralysis is on the left side. You notice that the foot is in a position of extreme equinus, but that there is little or no rigidity in the paralysed arm, also that the paralysed members are much smaller than the sound ones. You might be inclined from these facts to regard this case as one of "infantile paralysis." Such a diagnosis is excluded by the increase of myotatic irritability (which I now show you) on the paralysed side. I do not give such a favorable prognosis in this case as in the other, on account of the degree of atrophy present. This results from disuse, and therefore indicates considerable paralysis, with corresponding severity of lesion.

CASE 8.—This boy is 9 years old, and he was paralysed when he was two months old. You will notice extreme atrophy of the legs below the knees. The right leg is more atrophied than the left. At the same time one notices in connection with this fact that there is no deformity of the right foot, while the left one is in a position of extreme equino-varus. This is because paralysis is more complete on the right side, there being no unparalysed muscles to distort the foot. On the left side the calf muscles have escaped, and not being antagonised, have become permanently shortened, producing the equino-varus. Observe the extreme flexion of the limbs at the knee-joints. This is due to shortening of the hamstring muscles, owing to paralysis of the extensors of the knee.

Then you will notice also that sundry muscles in the right upper extremity are paralysed and atrophied, notably those of the shoulder girdle.

All the paralysed muscles are irresponsive to electric stimuli.

This case is an interesting instance of the marvellous power of adaptation possessed by the animal organism. (The patient walked backwards and forwards along the floor on hands and knees.) Having so many muscles paralysed, and having both legs permanently flexed at the knee, you would expect his powers of progression to be limited; but, as you observe, he manages to get along very well. (The patient then climbed into a high chair and seated himself.) You notice the extraordinary pads on the knees. They remind one of the heel, and they, indeed, constitute his heels.

CASE 9.—I will not spend long over this case. It is, like the last, one of infantile paralysis. There is also paralysis of the muscles about the right shoulder-joint. The right upper extremity, you will notice, is much smaller than the left, and not only are the muscles smaller, but doubtless the bones also. In cases of old infantile paralysis the bones and all the structures of the affected limb fail to grow properly.

CASE 10.—This is a case for diagnosis. The little girl is 8 years of age, and never has walked properly. You notice the child tends to walk on her toes like the children with spastic paraplegia. At the same time there is a certain amount of inco-ordination and tremor in the legs. The knee-jerk is distinctly exaggerated. There is also a tendency to inco-ordination in the upper extremity. (The patient picked up a small piece of paper.) This case does not quite conform to the ordinary ones of spastic paraplegia on account of this inco-ordination, and on account of the tremor, which is increased on voluntary effort. But you see there is a tendency for the child to walk on the toes, and that increased myotatic irritability is present.

CASE 11.—The last case I shall show you is one of old hemiplegia. The attack came on four years ago when the patient was forty-four years of age, and as there was no heart disease, the presumption is that it was due to syphilitic thrombosis, this view being further supported by the fact that he did not lose consciousness at the time, and by the deliberate manner in which the paralysis came on. At present the leg shows no evidence of paralysis. He lifts his toes up well, and does not swing the

leg round. If you look at the face you will notice that the right side is slightly paralysed. The right arm is weaker than the left. The speech has from the beginning been markedly affected.

Together with the hemiplegia and the aphasia he has hemianopsia. The lesion being in the left hemisphere, we get the failure of vision on the same half of each retina, *i. e.* the left half. This is the invariable rule, and the left half of each retina being affected, the right half of each field is dim. In this particular case the defect manifests itself chiefly in regard to colours. He can discern objects to his right fairly well, but he has no idea of their colour. (Patient was unable to tell the colour of an object held to his right, though he could dimly see the object; when held to the left he recognised the colour.)

The main points, then, of this case are hemiplegia coming on deliberately without loss of consciousness, affecting the right side of the face and the right arm, not so much the right leg; persistent aphasia and hemianopsia. The patient has not reached the age of arterial degeneration, and there is a history of syphilis. The diagnosis is, therefore, thrombosis secondary to syphilitic arteritis.

NOTES.

The Treatment of Chronic Heart Disease

by warm, medicated baths, according to the "Bad Nauheim" or "Schott system."—Since the presentation of the "Schott method" of treating heart disease by S. S. Cohen, many inquiries have been received concerning it. This extensive interest has probably been awakened because of the great frequency of cardiac affections and the questionable results met with by routine treatment, as compared with the brilliant results reported from Schott's method. Sir T. Grainger Stewart, in his opening remarks upon the Schott treatment before the British Medical Association at Carlisle in September, 1896, said: "Although the interest attaching to such a subject cannot compare with that of the announcement of the result of original research, yet in respect of practical importance it may deserve even more attention, and may in the end

prove of greater value to the practitioner, for, even as things stand at present, the management of cardiac troubles constitutes a responsible part of a physician's work, but as the frequency of preventable disease decreases, it will become of even greater moment." The *rationale* of the system has been asked for, and also explicit directions for giving the baths. At Bad Nauheim, treatment is usually begun with the baths, which are five in number. The waters contain large quantities of NaCl, Fe, and CO₂. The temperature is 90° to 92° F., which is lowered after immersion and during the patient's stay in the water to 85.5° F. The duration of the baths is six to eight minutes. They may be artificially prepared, and can be perfectly carried out in a hospital or private house. A porcelain tub is recommended. Treatment is begun with a quiet saline bath, and gradually increased to a turbulent sprudel bath or strombath. Robert Saundby gives the following directions:—To forty gallons of water, sodium chloride (NaCl) and calcium chloride (CaCl₂) with, later, sodium bicarbonate (NaHCO₃) and hydrochloric acid (HCl), are added in the quantities given by the following table:

Bath 1. NaCl 4 lbs.,	CaCl ₂ 6 oz.
" 2. NaCl 5 lbs.,	CaCl ₂ 8 oz.
" 3. NaCl 6 lbs.,	CaCl ₂ 10 oz.
NaHCO ₃ 6 oz.,	HCl 7 oz.
" 4. NaCl 7 lbs.,	CaCl ₂ 10 oz.
NaHCO ₃ 8 oz.,	HCl 12 oz.
" 5. NaCl 9 lbs.,	CaCl ₂ 11 oz.
NaHCO ₃ 1 lb.,	HCl 1½ lbs.
" 6. NaCl 11 lbs.,	CaCl ₂ 12 oz.
NaHCO ₃ 1½ lbs.,	HCl 2½ lbs.

He uses only the first four, beginning with a quiet saline bath. W. Bezley Thorne uses the following proportions of sodium bicarbonate and hydrochloric acid:

Mild—NaHCO₃ ½ lb., HCl (25 per cent.) ¾ lb.

Medium—NaHCO₃ 1 lb., HCl 1½ lbs.

Strong (Sprudel strength)—

NaHCO₃ 2 lbs., HCl 3 lbs.

He says, "For the production of carbonic gas effervescence the action of HCl and NaHCO₃ is relied upon. The commercial articles answer all purposes. Two ways of employing the reagents are suggested. The one is calculated to induce slow and gradual, the other rapid, almost immediate effervescence. In the case of the first the various

salts, including the requisite proportion of NaHCO_3 , having been dissolved, a bottle containing the acid is laid at the bottom of the tub, and the stopper having been withdrawn, it is moved about from time to time. The bath will be ready in two to three hours. For the more rapid production of effervescence the stopper of the bottle containing the acid is loosened, but retained in position, the bottle having then been inverted and lowered until the mouth is just below the surface of the water; the stopper is then withdrawn, and the bottle is moved about so as to diffuse a layer of acid as uniformly as possible over the surface of the bath. By this means the bath will be prepared in about five minutes."

Sir T. Grainger Stewart recommends Sandow's tablets or powders to produce effervescence. He says one powder is equal to a Sprudel, and two to a "Strom" bath. He has seen the most striking results while a patient was immersed, both as to diminution of the area of cardiac dulness and as to reduction of the frequency of the pulse. He made a series of experiments with ordinary Edinburgh water at 95°F. , which failed to show the slightest effect on the heart and pulse; mere saline baths were equally resultless, but when combined with Sandow's powders, had the same effect as had been observed at Bad Nauheim. We have used the saline baths upon two patients of Dr. S. Solis-Cohen at the Polyclinic without any pulse-reduction whatever; while, with one of these patients, the effervescent baths (Nos. 3 and 4) of Saundby had immediate effect. The baths alone are often beneficial, as the exercises sometimes make an irritable heart worse.

The following account of the action of the baths is condensed from Stewart. The skin is reddened, due to the dilatation of vessels; the pulse becomes slower and of more regular rhythm. The peripheral resistance is diminished, rendering the work of the overtaxed heart lighter. Now the natural physiologic effect of diminished peripheral resistance would be to quicken the heart's action and cause a fall of arterial pressure. How is it that we get a slowing of the heart's action and a rise of pressure? The explanation may lie partly in the circumstance that the heart has been acting irregularly and too quickly in consequence of its unfitness to meet the demands made upon it, and that when it is relieved of a portion of the blood

which was overfilling its chambers it becomes equal to its work, performs it more slowly and efficiently. It may also be true that with the attraction of blood to the peripheral vessels the influx of venous blood to the heart is increased, the removal being diminished, and so long as this influx does not rise beyond the heart's power of propulsion, the action becomes more vigorous, and the arterial tension is raised, the balance of circulation being improved. He is not prepared to exclude the influence of baths largely upon the nerves of the skin acting upon the innervation of heart and vessels, as the simple warm bath does not bring about the same result.

The theory adopted by Dr. Schott is that the baths and exercises act upon the heart through the nervous system by evoking a reflex influence which stimulates the action of the cardio-inhibitory or regulator nerves, thus slowing and strengthening the pulse, and though some of his friends in England take a different view, he adheres to this explanation.

Thorne's theory is that the baths and exercises dilate the arteries of the muscles and those of the periphery generally, and thus relieve the heart from backward pressure. This view is supported by Sir William Broadbent, and his son, Dr. John Broadbent, and was likewise upheld by Dr. Cohen in his discussion before the County Medical Society. Saundby says this view is not consistent with the pulse tracings, nor with the physiologic law that the heart beats vary inversely with the blood-pressure. If the effect of baths and exercises be to diminish blood-pressure, the heart's action, following Marey's law, would be quickened; all are agreed that the pulse is slowed.

Saundby believes the principal effect is on the peripheral circulation (thus differing with Schott), but agrees with him that the blood-pressure is raised, and, according to Marey's law, the heart's action is slowed. His theory is that the beneficial effects of treatment are due to the fact that the extra work imposed upon the heart strengthens and develops its muscular wall, just as any muscle is strengthened and developed by well-regulated systematic exercise, and not to any hypothetic reflex nervous effect upon the heart.

Thorne says, "The effect on the peripheral vessels is to increase their carrying power; a glowing sense of warmth is experienced in the

extremities and on the surface of the body generally; the veins are stimulated to a similar activity; in fact, the general vascular capacity, systemic and pulmonary, is increased, and without loss of blood the relief of a general bleeding is afforded to an overloaded and labouring heart."

Sir Philip C. Smyly says: "There will ever be a feeling against this treatment until it is clearly seen and believed that—(1) The movements relieve the back pressure of the heart. (2) That the diminution in the size of the heart is due to the absence of an excess of blood in its cavity. (3) That this is attained by there being more room in the arteries. (4) That the heart muscle gains strength by having room to contract. (5) That the contraction being more complete, it takes a longer time, thus making the pulse slower and at the same time fuller. (6) Being able to send on more blood, it is ready to receive more, and thus removes venous congestion. (7) The strength gained by the heart is due to the freedom to contract fully.

Philadelphia Polyclinic, May 29th, 1897.

SPEAKING of *psoriasis* Dr. Cantrell asserted his firm belief in the internal treatment by oil of copaiba, feeling certain that in the dose of five minims, thrice daily, good results would accrue; but this should be increased as high as ten or even twenty minims if the smaller dose should prove insufficient.

Philadelphia Polyclinic, May 22nd, 1897.

The Latest in Roentgen Photographs.—

Kümmel has traced with the Roentgen ray the course of the Murphy button through the organism. Syndactylia in a child was successfully treated with information derived from paternal photographs. Stechow thinks the importance of this photography in army surgery cannot be too highly estimated. Levy Dorn locates foreign bodies with extreme precision by passing a needle around the part, held behind the fluorescent screen. The two points at which the shadow of the needle coincides with the shadow of the foreign body are marked on the skin. Repeating this in two or three different positions, the position of the foreign body can be found exactly. Another method is by moving the light slightly, and thus taking stereoscopic photographs. Max Levy reports that it is

now possible to take photographs of the thorax and pelvis with an exposure of only thirty to sixty seconds. The time has been thus shortened by obtaining a more complete vacuum, laying a supplementary screen over the sensitive plate, and by using specially prepared plates with the sensitive film twice and four times the usual thickness.

Congress of Surgery, Berlin, April.

Unsuspected Causes of Rebellious Neuralgias.—When sciatica or neuralgia persists for a long while, uninfluenced by treatment, some osseous lesion of the skeleton may prove the cause. Moutard-Martin describes a case which lasted two years before it was traced to an iliac osteo-sarcoma. Siredey and Grognot have likewise observed Pott's disease develop after months of a persistent neuralgia.—*Presse Méd.*, Feb. 27th.

REVIEW.

Surgical Diseases of the Ovaries and Fallopian Tubes, including Tubal Pregnancy. By J. BLAND SUTTON, F.R.C.S., Assistant Surgeon to the Middlesex Hospital for Women, Surgeon to the Chelsea Hospital for Women. New and enlarged edition. London: Cassell & Co., Ltd. (Demy 8vo., pp. 444, 146 Illustrations. 21s.)

The new edition of this popular work contains twenty-seven more drawings than in the first edition, and it has the great advantage of being printed in type which is larger and clearer than in the first appearance of the work. We are told in the preface by the author that his object is "to enable deduction from observation to replace hypothesis and speculation;" after the reader of this work has duly appreciated the immense number of facts set forth by the author, and has given due weight to the individuality with which the lessons to be deduced from the facts are logically discussed, we are confident in asserting that that object will be found to have been attained. Apart from the clinical lessons to be derived from the perusal of this volume, much pathological knowledge is admirably and lucidly brought forward. No one can read this book without fully appreciating the analytical power displayed in the treatment of the

questions discussed, and we congratulate the author in possessing the capacity to so thoroughly sift difficult details, whilst still retaining the interest of his reader—a result due to a happy knack of dealing with figures and percentages intelligently and clearly. What appears to the author to be still obscure is boldly stated to be so, and in place of questionable guesses we are met with the plain statement that reliable data are not to hand on which to form an opinion. The chapter on menstruation ends up with the words that “the cause of menstruation remains as obscure as ever.” Again, speaking of warty ovaries, the author says, on page 95, “of the cause and significance of these hard wart-like bodies I am quite ignorant.” In the chapter on menstruation the opinions of Heape on the menstruation of the Entellus monkey are quoted; the author considers that these bear out in the main the contention that the menstrual changes are limited to shedding of the uterine epithelium, and that the tubal epithelium takes no part in this extraordinary function. In the chapter dealing with corpora lutea a calcified corpus luteum is figured and described.

Concerning the question of primary ovarian and primary abdominal pregnancy, the author denies that there is any evidence of their existence, and on page 35 observes that “until a specimen is forthcoming with an early embryo, and its membranes contained in a sac inside the ovary, we need not trouble to discuss ovarian pregnancy.” Salpingocele and its treatment receive an adequate notice in Chapter IV, and the condition is well illustrated in fig. 18. On page 49 there is an excellent diagram representing the cyst regions of the ovary, and the classification of cystic growths according to the region of origin is well and clearly explained. The importance of ovarian dermoids is sufficiently recognised by the fact that Chapters VIII, IX, and X are devoted to their consideration. Axial rotation is fully discussed, the author holding that acute torsion is more frequently seen in tumours of medium size. Acute torsion does occur in small cysts, but it is in the small tumours, especially dermoids, that slow torsion takes place. In Chapter XXVII, on neoplasms of the Fallopian tube, there is an interesting account of a true myoma of the tube removed by operation. This case is interesting as being the only example which has come under the notice of the author,

and is an exceedingly valuable addition to the literature on the subject. In the same chapter the author states “it is quite certain that adenomata of an interesting and characteristic type occur primarily in the tube.” Although a celebrated German pathologist does not admit that glands are present in the structure of the tubal mucous membrane, facts are advanced by the author showing that the folds in the tubal mucous membrane are glands. As is justly observed, if the Fallopian tube contain no glands it is impossible that it can give rise to a primary adenoma.

In this admirable work it is difficult to point to any part as being of special interest or importance, but from the practical point of view probably one would choose Part III, devoted to tubal pregnancy, as being the most useful portion, to which part also some new illustrations are added. On page 245 Mr. Bland Sutton gives his opinion on the question of the connection of chronic salpingitis and tubal pregnancy in no uncertain manner, and he adds, “as a matter of fact the evidence now indicates that a healthy Fallopian tube is more liable to become gravid than one which has been inflamed;” the chapter concluding with the statement that “the chief cause of tubal pregnancy remains undetected.” Part IV treats of diseases of the pelvic peritoneum, and though want of space alone prevents us doing this portion of the work full justice, it is impossible in a notice of this book to omit mentioning the remarkably clever description of anterior parametritis. The explanation of this unusual variety of pelvic cellulitis is excellently illustrated by a figure showing a sagittal section of the parts involved, from a specimen in the museum of the Royal College of Surgeons. The fifth and concluding part of this volume treats of the methods of performing operations for ovarian and tubal disease. Much difference of opinion, of course, must be expected concerning these operations; but the clearness of the directions, and the careful way in which the details are explained, must render this part of the greatest use to the medical profession. It is impossible in a short space to do more than indicate the excellence of this remarkable work, and the more books we have written to replace hypothesis and speculation by deduction from observation, the more will medical literature be enriched by volumes of real use to the every-day worker.

THE CLINICAL JOURNAL.

WEDNESDAY, JUNE 23, 1897.

A CLINICAL DEMONSTRATION ON CHRONIC ENLARGEMENTS OF GLANDS.

At St. Bartholomew's Hospital, March 5th, 1897, by
HENRY T. BUTLIN, F.R.C.S., D.C.L.,
Surgeon to the Hospital.

GENTLEMEN,—When I decided to give two lectures on affections of the glands,—one on the treatment of tuberculous glands, and the other on the diagnosis of chronic glandular diseases,—I intended to restrict the second within very narrow limits; but the more I came to consider the matter, the more difficult I found it to do this. It opens up so many large and important questions that it seems impossible to consider the diagnosis of one or two of these chronic affections of the glands, even of the neck, without going deeper into the subject of enlargement of glands generally.

We must first consider what different enlargements of the glands may be met with, and to this end the following rough classification may be useful.

- I. Inflammatory (chronic).
- II. Infective (examples: tubercle and syphilis).
- III. Tumours—1. Innocent (lymphoma).
 - 2. Malignant.
 - Primary—*a.* Lymphadenoma.
b. Lymphosarcoma.
 - Secondary—*a.* Sarcoma.
b. Carcinoma.

A chronic inflammatory affection of a gland may be the sequel of an acute affection. An acute inflammation in any part of the body ought to get well if the patient is in good condition, treated, and the cause of the inflammation is removed; but if the patient is not healthy, or the cause of the inflammation is not removed, the part may remain in a state of chronic inflammation. A large number of the chronic enlargements of

the glands are tuberculous, or due to some general diathesis, not a mere sequel of an acute inflammation. If a patient is brought to me with a chronic enlargement of a gland, and I am asked whether that is simply a chronic inflammation or some other disease, I reply that it is very unlikely a person should have such a chronic enlargement unless there be some diathesis to account for it, and most likely the tuberculous diathesis.

I next come to infective tumours, and of these I spoke the other day. Two good examples of this class are tubercle and syphilis.

Next are the new growths,—and here difficulty begins. I divide them into innocent and malignant. As an example of innocent growth I mention lymphoma,—a hypertrophy or hyperplasia of an ordinary lymphatic gland.

Primary malignant growths come next, and these I divide into lymphadenoma and lymphosarcoma. Secondary malignant growths are of two kinds—sarcoma and carcinoma.

Now on some of these conditions we are quite agreed; we know perfectly well what we are talking about with regard to them. We know that a gland which has been inflamed may, instead of subsiding, remain enlarged, though the active inflammation has passed off. This we speak of as chronic inflammation of the gland. I do not think the term is quite a correct one, because it does not exactly express the truth. In most cases there is no actual inflammation. The inflammation which caused the enlargement has passed off, but the products of inflammation have not been removed, and the gland remains enlarged. As examples I may mention that yesterday and to-day I saw young ladies aged twelve and fourteen years respectively. Each had some adenoid vegetations, and each had enlarged glands behind the jaw on either side. These glands were not tender, but were about twice the normal size, and there were no signs of inflammation about them. In one case the mother knew of the enlargement, but in the other the condition had not been noticed. The glands had been "set alight," as it were, by some inflammatory

trouble in connection with adenoid vegetations, and instead of going down they remained enlarged. A catarrhal inflammation of the adenoids might cause the glands to swell up again, and thus they might be regarded as in a chronic state of inflammation.

As to the infective tumours—tubercle and syphilis,—we are generally quite clear as to the nature of the disease and the diagnosis. In tubercle, for instance, as I have shown you, the diagnosis is made from the general characters of the glands, from the history of the patient, from the family history; then, for confirmation, the glands are examined, and tubercle found in them. I show you an enlarged diagram of a tubercle in a lymphatic gland. Tubercle bacilli are not always demonstrable in these cases; still, the diagnosis of a case of ordinary tubercle of lymphatic glands is generally to be made with positive accuracy. Syphilis is usually, particularly when the disease is secondary, easy to diagnose.

We now come to new growths. Those of you who heard the discussion that took place yesterday at "Consultations" upon some of these cases here* may have noticed that one spoke of lymphadenoma, another talked of lymphosarcoma, and a third of lymphoma. My colleague, who used the term "lymphoma," said he was not quite sure what was meant by the term, and I agree with him that the name is used in a very wide sense. The conception of lymphoma is this:—Take a single lymphatic gland, and suppose that irritation or chronic congestion, or some other cause, sets it growing as a lipoma or fibroma is set growing in fat or fibrous tissue. Suppose there are no limits to its growth; but that its structure resembles the structure of a normal lymphatic gland, and that it is still contained in a capsule, and even retains its ordinary shape, and that it does not infect the other glands in its vicinity. I have often wondered whether such a disease exists. Some years ago I thought I had seen a case of true lymphoma. A gentleman about forty years of age consulted me. He was suffering from a large gland in the neck; it was oval in shape, and situated beneath the sternomastoid muscle. The patient did not know what had caused it, and there was no other enlarged

gland to be felt in his neck or body. It steadily increased in size, and at one point I thought it was beginning to break down. I advised that it should be removed. I performed the operation, and found that it was not breaking down. It looked much like an ordinary lymphatic gland, but pale and more waxy. It was as large as a hen's egg, and was enclosed in a capsule, out of which it shelled like an ordinary fatty tumour. I said to myself, "that seems the best illustration I have seen of my conception of true lymphoma, for it bears almost the same relation to a lymphatic gland as a fatty tumour does to normal fat." The structure of the tumour differed little from that of a lymphatic gland, and there was no tubercle. Within a few months the patient came back with several other glands enlarged on the same side of the neck, and as these were sufficiently moveable I cut down upon them, and found some of them contained caseous material, and some of them were breaking down. Then I began to wonder whether the disease was tuberculous, but it was not. The glands were examined, and they presented the same structure as before. After the second operation the disease extended, and in course of time a large gland formed in the neck, causing paralysis of the facial nerve; and in one and a half years the patient died from this affection of the glands, which implicated other glands of his body. Whether he had any other secondary growths I cannot say. Some people speak of every enlargement of a lymphatic gland as lymphoma. I do not like the term, because it seems to imply such a disease as I have given you a conception of, like lipoma or fibroma.

Next we come to malignant disease. Take first the *secondary* forms. I show you photographs of a woman with enlarged lymphatic glands in the neck secondary to an affection of the larynx, which was comparatively slight. I show you also the picture of a mass of sarcomatous glands. They were supposed to be secondary, but no one had the least doubt about the nature of the disease. As regards the diagnosis of such glands as that woman presents, there is no particular difficulty as a rule. The glands are exceedingly hard, are generally very deep-seated, and become fixed at a very early period. They often press upon and involve the nerves of the neck, so that paralysis of the sympathetic nerve or of the recurrent

* Three cases of lymphadenoma, which were under the care of my colleague, Mr. Willett, were exhibited by his kind permission.

laryngeal nerve is a very common result of the progress of the disease. Even when the primary disease itself, owing to its being small or hidden, is not discovered, the characters of the glands are so significant that at our Thursday consultations you will have noticed we generally are quite agreed on the nature of these secondary glandular diseases, though we may not have discovered the primary disease.

But when I come to *primary* forms of malignant disease of the glands, I realise that it is a subject which presents very great difficulties. Of the patients whom I send round for your inspection, we came to the conclusion that at least one of them is suffering from lymphadenoma, another name for which is Hodgkin's disease, because a Dr. Hodgkin—who ought to have been physician to Guy's Hospital—but never was so—first gave a very excellent description of some of these cases. For a long time past the patient I refer to has had "a lump" in his neck, and you will notice on examination that it is made up of a large number of tumours about the size of a nut or pigeon's egg, making one huge mass. You can feel that they are like big lymphatic glands, which can be freely moved one upon the other, and the whole mass can apparently be moved on the deeper parts. There is no sign of softening at any spot, or of breaking down, so that the chances are it is not an inflammatory affection or an infective tumour, but belongs to the lymphadenomatous diseases. Some enlargement of the axillary glands can also be made out. When we come to diagnose these diseases we often find ourselves in very great difficulty, and yet it is of great importance that a correct diagnosis should be made. If I show my class in the ward a case of Hodgkin's disease, and ask in what parts of the body the tumours may occur, I get very correct answers; also to my query as to whether the disease affects any of the internal organs. When I ask what are the microscopical characters of the disease I am told that they do not differ much from the microscopical characters of an ordinary enlarged gland. On this point I am obliged to differ from my class. Yet I cannot deny that there are some cases of lymphadenoma in which the microscopical structure of the disease is very much like that of an ordinary enlarged gland. Before I became registrar to this hospital, I was registrar

at the Children's Hospital, Great Ormond Street. Into that hospital a boy was admitted with enlarged glands in the neck, which had been present a year or more. The glands showed no sign of breaking down, and there was no softening of them, such as one finds in suppurating or tuberculous glands. The glands in both axillæ were enlarged, and I believe those also in the groin. There were certainly symptoms pointing to enlargement of the mediastinal glands. He was under the care of Dr. Dickinson, and was kept in the hospital for a considerable time, during which he gradually became weaker, but none of the glands broke down. At length he had epistaxis, which very greatly reduced his strength, and shortly afterwards he died. I made the post-mortem examination, and found enlarged glands in the abdomen as well as in the mediastina, and some enlargement of the liver and spleen. I made a careful microscopical examination of the glands, and I must confess I could not distinguish any very great difference in the characters of these lymphadenomatous glands and those of an ordinary tolerably healthy subject. So that I suppose there is at least one form of lymphadenoma of which the microscopical characters are very much like those of an ordinary lymphatic gland. On the other hand, there is a class of cases to which we give exactly the same name, which run much the same course, but which present a very different structure on microscopical examination. I will try to describe a typical instance. A young lady was brought to me some time ago with a number of enlarged glands on both sides of the neck. They differed from those you have felt to-day, because, although some of them were very moveable and not very hard, others formed large masses which were adherent to the structures in the neck. She had also scars in the neck, as some glands had already been removed. One of the masses was adherent to the skin; the skin was reddened at the spot, and her doctor brought her to me on the question of diagnosis and operation. In her case a very important question was raised. She was twenty-three years of age, and was engaged to be married. Her *fiancé* had come over from abroad to see her, and probably to be married to her. Her father was very anxious for the marriage to take place, and the following questions were put to me:—(1) What is the nature of the disease? (2) Should another operation be performed? (3)

Should she go on a sea voyage? (4) Should she be married? One of the views was that the disease was tuberculous, and her father, who had sufficient education to know what tuberculous disease meant, said, "If it is nothing but that she may get well, and it will do her no harm to get married." I examined her, and found in the neck huge masses, firmly adherent. Therefore operation was out of the question. I came to the conclusion absolutely that the affection could not be tuberculous, particularly after looking at some sections under the microscope of the glands which had formerly been removed, showing appearances identical with this enlarged picture I show you, taken from the Pathological Society's 'Transactions.' It was a hard form of lymphadenoma, and naturally I urged strongly that she should not go for a sea voyage, and said that it would be very wrong for her to get married. I do not know whether my advice was taken. Some of the glands which had been removed had been sent to London for examination and report. They had been examined by a very competent microscopist, who was not familiar, however, with this form of lymphadenoma, and expected to find a structure resembling that of an enlarged lymphatic gland. Not finding this structure, he thought the disease was most likely tuberculous, although no tubercle could be demonstrated. The diagnosis of tubercle was made, and on that the father founded his scheme. I think this peculiar structure in the picture I show you, reproduced from Dr. Greenfield's illustration in the Pathological Society's 'Transactions,' is pathognomonic of one form of lymphadenoma. But I believe there are two quite different diseases (pathologically) included under the same name, or that the same disease presents very different structural characters under certain circumstances.

Another disease of glands of which I am very ignorant is lymphosarcoma. When, in other organs of the body, such as the testes or tonsils, we meet with a tumour which presents, not the ordinary structure of a round-celled sarcoma, but a reticulated structure like that of a lymphatic gland, and having cells like those of a lymphatic gland, we have been in the habit, for many years, of speaking of it as lymphosarcoma, and when that kind of disease occurs in the lymphatic glands as a primary disease we speak of it as lympho-

sarcoma, and I have in my mind an illustrative case. Some years ago Mr. Willett had under his care a boy of about seven or eight years of age, who had a large mass of glands on one side of the neck, which had formed in a few weeks. It grew so fast that almost day by day we could mark its progress. But there were no other enlarged glands in any part of the body. In course of time this mass broke down and sloughed, and in a very few weeks the little fellow died from the sloughing and rapid growth of the disease in his neck. I do not quite recollect, but I believe some secondary growths were found, post mortem, in other parts of his body. The microscopical characters were very much like those represented on the drawings I send round. You will see that the structure does not differ materially from that of some of the forms of lymphadenoma, and I am not prepared to say that this deserves to be classed as a separate disease.

As to the nature of lymphadenoma I am in the greatest doubt. It evidently is not a very infective disease, like tubercle; if it is infective, the infective material has not been discovered. It differs in some important respects from any kind of real new growth, and I hope a clue to the nature of malignant disease may be found in the more careful study of these lymphadenomatous glands. In the first place, in some patients it runs an exceedingly slow course, and the glandular tumours vary considerably in size from time to time. A man was under my care with enlarged glands at the West London Hospital. Five or six years afterwards, when I was in the out-patient room here one day, he turned up unexpectedly and told me he had been under my care so many years previously. I had diagnosed his disease as lymphadenoma, and the subsequent diagnosis was the same. Some months afterwards I operated upon him to take the glands away. I took out a number of them with ease, but found that others involved surrounding structures, the great vessels and nerves. There was no suppuration or caseation, but I could not remove the whole of them. The patient made a good recovery. The sections bore the characteristic appearances of lymphadenoma, so that I had no doubt about the diagnosis. Two or three years later I saw that patient, whose disease had lasted certainly for ten years, and sometimes the glands were much smaller than at others. There are few

cases of real malignant disease in which the patient lasts for ten years, and there are still fewer instances in which the disease sometimes becomes very much smaller and then swells out again, sometimes leaving the patient comparatively well for a time. In the second place, the disease is often singularly amenable to the influence of arsenic. I had two patients under my care in the out-patient room at the same time, both of them suffering from lymphadenoma. I put them on liquor arsenicalis, beginning with 3 minims three times a day after meals; at the end of the first week it was increased to 4 minims, and so on, until at length to 13 and 14 minims three times a day. Under this the glands disappeared as if by magic. You may have seen a similar thing happen in a patient who was under my care in Sitwell Ward a few months ago. I do not say that arsenic is an infallible cure for lymphadenoma, or that it has a decided effect upon every case of lymphadenoma, but its influence on some cases its undoubted and striking. Here, again, we have a suggestion that there are at least two forms of the disease—that in which arsenic acts, and that or those in which it wholly fails. I know no real malignant disease which is influenced by any known medicine to that extent.

Judging, then, from the course which the disease sometimes pursues, from the different effect which medicine, and especially a certain medicine, produces upon it, I cannot help thinking it is some disease midway between infective tumours on the one hand and true malignant disease on the other; and I hope that during the next few years a careful study of these primary glandular affections may lead to a very great increase in our knowledge of lymphatic glandular diseases on the one hand, and to a much better knowledge of the real nature of true tumour diseases than we at present possess.

In conclusion, let me beg you to regard this lecture rather as suggestive than definitive; for I am obliged to confess that I am lecturing on a subject of which I am very ignorant, yet perhaps not more ignorant than others are, since the whole subject is in a very confused condition. You will see that I have not attempted to deal with the enlargements of glands which are associated with great enlargement of the spleen and a large increase in the proportion of the white corpuscles of the blood. Yet this disease, again must be very closely allied to some conditions of lymphadenoma.

I can only hope my lecture may induce some of you—if not now, by-and-by—to take up the study of these curious affections of the glands. I am sure you will be repaid by the interest of the study so far as it refers to the glands alone. And I am almost equally sure that the clue to the real nature of some at least of the malignant tumours of other organs and tissues is to be found in the discovery of the nature and relations of these glandular diseases.

A CLINICAL LECTURE

ON A

CASE OF PERNICIOUS ANÆMIA.

Delivered in the Western Infirmary, Glasgow,

By C. O. HAWTHORNE, M.B., F.F.P.S.G.,

Assistant Physician to the Infirmary, and Lecturer on
Materia Medica and Therapeutics, Queen Margaret
College, University of Glasgow.

GENTLEMEN,—The case which I wish to review with you to-day is that of the young woman J. D—, æt. 25, whom we saw for the first time on the morning following her admission, and who unfortunately died five days after entering the hospital. You will remember the special difficulties of the first examination, due to the exceedingly incomplete history supplied to us. She herself was manifestly very ill, and could say no more than that her health had been failing for some time, and that she had been in bed for a fortnight, during which time she had been troubled with diarrhoea; and the friend who accompanied her was unable to add any further particulars. That her illness was a serious one was obvious without any detailed examination. It had been necessary to carry her into the ward on account of her weak condition, and both her physical and mental powers were manifestly greatly prostrated. Further, her illness was of a febrile character. The pulse numbered 130, and the temperature, which on admission was 100·8°, had now risen to 102° F. Yet there were neither symptoms nor physical signs to suggest a local cause of this febrile condition. She had no pain, no cough or other discomfort

except "weakness," and the usual examination of the thorax and abdomen left us equally without suggestion or guidance. The febrile condition had, however, to be explained. As I remarked to you at the time, a high temperature without definite symptoms or physical signs to explain it, compelled us to think of the possibility of one of the specific fevers. The fact that she had been ill for at least a fortnight, the absence of anything like an acute onset, and the absence also of any cutaneous eruption, rendered the diagnosis of any of these very unlikely, except possibly one, viz. enteric fever. As you know, it is quite possible for this disease in particular to develop very insidiously, and for the patient to have no localising symptoms and no eruption on the skin, the highly suggestive "rose spots" being not infrequently very few in number, or even altogether absent. The presence of diarrhœa also was a fact which, under the circumstances, compelled us to think of enteric fever. It is a disease which always occurs to the mind of the physician when with diarrhœa there are high temperatures for which no adequate explanation can be discovered. In the present case there was a strong probability that the woman had been ill for many weeks; and though enteric fever may run a very protracted course, it is unusual for its febrile manifestations to persist for more than three weeks or so. Further, we had no positive evidences in favour of enteric fever—no abdominal distension or tenderness, no "rose spots" on the skin, no enlargement of the spleen; and a stool passed by the patient after admission, though loose, had not the dirty yellowish colour or disagreeable odour which we expect in enteric fever. The tongue was no doubt exceedingly dry, and covered with a dark brownish crust, and the pupils were dilated; but these conditions might be results of a continued high temperature, and were certainly not sufficient in themselves to support, or even to strongly suggest, a diagnosis of enteric fever. We perhaps could not at this point definitely exclude such a diagnosis, but the balance of probabilities seemed against it. Another suggestion which arises when there is persistent fever without physical signs indicating the existence of a definite disease is the suggestion of tubercle. There is no doubt that tubercle may exist, and produce high temperatures, lasting for a considerable time, without betraying itself by any

distinctive localising symptoms or physical signs; and diarrhœa is not an uncommon manifestation under such circumstances. The case as it grew up before us at the bedside might, as I remarked at the time, be possibly one of this nature. So far, then, we could not dogmatise beyond this point. Here was a young woman seriously ill with some febrile disorder which had probably been present for several weeks. The absence of physical signs left us without any explanation of the fever so far as the thoracic and abdominal viscera were concerned, and the mere trace of albumen present in the urine might well be a result of the febrile disturbance. The duration of the illness, and the freedom of the skin from eruption, excluded the great majority of the specific fevers. Under these circumstances the possibility of the high temperatures being due to enteric fever or to tubercle had necessarily to be considered; and though neither could be definitely excluded, there was no positive evidence in support of either diagnosis. It seemed highly probable at this point that our attempts to reach a distinct diagnosis would end in failure.

Now you will remember that in our examination of the patient we were struck with the fact that she was very anæmic, and that her pulse was very weak. These conditions might, no doubt, to a considerable extent be secondary consequences of protracted high temperature, but they impressed me as being unusually severe. And I was rather disposed to think that the skin was not merely pale, but that it presented a faint yellowish tint—a yellowish tint which was not due to jaundice, for the conjunctivæ as we inspected them were quite clear. We now examined the fundus oculi with the ophthalmoscope, and by doing so we were immediately introduced to a fact of great significance in connection with the diagnosis of the case—for in each fundus were numerous and extensive hæmorrhages. In the light of this fact a diagnosis of enteric fever or tuberculosis distinctly receded from our minds. We do not expect retinal hæmorrhages in either of these conditions; and, on the other hand, the presence of these hæmorrhages strongly suggests one or other of the diseases associated with extremely grave alterations in the quality of the blood—one of these diseases being pernicious anæmia. I emphasise again the illustration which this case affords of the great value of the ophthalmoscope

to the physician. Here we were dealing with a serious case of illness, and yet were almost completely in the dark as to its true nature, in spite of the fact that we had exhausted the usual methods of physical examination, and had fully considered such history of the case as was at the time accessible. But as soon as ever the ophthalmoscope is used we get a fact which is most direct and forcible in its significance, and our difficulties so far as the diagnosis is concerned largely disappear, for we know that in a certain limited group of diseases,—of which pernicious anæmia is one,—evidences of extreme anæmia are apt to be associated with retinal hæmorrhages, and with outbursts of pyrexia occurring without discoverable cause. No doubt, as I have just indicated, there are other diseases more or less closely allied, at least clinically, to pernicious anæmia, in which the same combination of events may obtain; but the diagnosis of any of these could be excluded from the present case for reasons I shall remind you of in a moment or two. My point just now is that by the use of the ophthalmoscope we were at once put upon the right lines for obtaining a specific and confident diagnosis when the more ordinary methods of examination had left us hesitating and uncertain.

With the suspicion of pernicious anæmia before us it naturally became of the first importance to examine the blood. That examination demonstrated, as we expected, a condition of marked anæmia. The red corpuscles numbered only 10 per cent., and the hæmoglobin was present only to the extent of 16 per cent. of the normal. This clearly proved extreme anæmia; but it did more than this, for whilst in most cases of simple anæmia the reduction of the hæmoglobin is considerable, the corpuscular defect is much less marked. But in pernicious anæmia it would appear as if there was breaking up of the red corpuscles, with escape of some of the colouring matter into the blood serum, for it is not infrequent to find that the reduction of the corpuscular elements is more marked than the deficiency of the colouring matter as this is estimated by the hæmoglobinometer. Further, we noted that the red corpuscles were of various sizes, and that they presented many varieties of form—a condition known as poikilocytosis, and frequently observed as a marked feature in the blood of patients suffering from pernicious anæmia. The white corpuscles

of the blood appeared to be present in fairly normal proportions—a fact of considerable importance, as will appear immediately. The condition of the blood, in short, distinctly supported the diagnosis of pernicious anæmia.

The history of the case may now be referred to. It is more completely in our possession since the death of the patient, as we have seen the practitioner who was in attendance on the patient before her admission. He informs us that she was the mother of two children, the first born in January, 1895, and the second in August, 1896, three months before admission. During the last month of the second pregnancy she suffered from some vomiting, diarrhœa, and swelling of the feet, but otherwise the pregnancy, labour, and puerperal period were quite normal; and there were no post-partum or other hæmorrhages. Since the birth of the child the patient has had considerable domestic anxiety, and probably has not obtained adequate food, and there has been gradual failure of her strength and health, but without recognised loss of flesh. There has, therefore, been no distinct event in the patient's history, such as unduly frequent child-bearing or conspicuous loss of blood, to account for the anæmia. It must therefore be placed, not among the secondary but with the primary or idiopathic anæmias; and one of these is pernicious anæmia. Further, it has been distinctly recognised that a period of special mental anxiety frequently precedes the onset of this disease, and that the disease itself, though marked by extreme weakness, is often unattended by any considerable loss of flesh. So that the history of the case can be claimed in support of the diagnosis to which our examination of the patient had already inclined us.

I referred a moment ago to certain other diseases of which the evidences are, as in pernicious anæmia, extreme reduction of the quality of the blood, retinal hæmorrhages, and, at times, febrile temperatures. Understand, I do not say that either in these or pernicious anæmia itself disturbance of the temperature is a constant feature. In all these diseases the temperature may be quite normal, but in all of them also there are apt to occur outbreaks of pyrexia. The diseases I am now referring to are leucocythæmia, Hodgkin's disease, and splenic anæmia. We had no difficulty in excluding these from our present diagnosis.

The case was not one of leucocythæmia, because there was no enlargement of the spleen, and no aggravated increase of the white corpuscles of the blood—facts which are leading clinical features of that disease. The non-enlargement of the spleen excluded splenic anæmia. In Hodgkin's disease we expect to find one or more of the superficial groups of lymphatic glands enlarged. Such enlargement is frequently the earliest observed fact in Hodgkin's disease, and its absence from this case placed that disease without the diagnosis. Cases of internal cancer, for example cancer of the stomach, have on more than one occasion been diagnosed as pernicious anæmia. But the age of the patient, the absence of any decided loss of flesh, the severity of the anæmia, the presence of retinal hæmorrhages, and the marked pyrexia, formed in the present case a combination of facts strongly against cancer. One other disease may be mentioned of which marked prostration without physical signs to account for it is one of the leading signs—I mean Addison's disease. In that disease, as you know, there is usually a bronzed discoloration of the skin. But this is not always present, and extreme prostration, with weakness of the pulse, and vomiting, may be the only positive evidences. It is noted, too, that in Addison's disease, as in our present patient, the loss of strength is out of proportion to the loss of flesh. But the severity of the anæmia in our patient is quite unusual in Addison's disease. Further, we do not get in this disease retinal hæmorrhages, and the temperature is rather subnormal than febrile. Hence we did not seriously consider Addison's disease as a possible diagnosis in the case. We were able, indeed, to exclude with considerable confidence both this and the other diseases I have mentioned, and to remain true to the diagnosis suggested by the history and examination of the patient, viz. that of pernicious anæmia.

The subsequent course of events only too faithfully confirmed our diagnosis. The patient became more and more prostrate, the pulse more frequent and weaker, and diarrhœa and vomiting very troublesome. These circumstances were also of grave omen. Very soon the aspect of the case became even more serious, and I impressed upon you the prognostic significance of the facts, even apart from the specific diagnosis of the case. When, as in this case, we find that a sudden and considerable fall of temperature is accompanied by increased

rapidity and weakness of the pulse, we know there is immediate and pressing danger of collapse. A decided increase in the rapidity of the respirations—the rate in our patient reached 55—is also another ground for serious anxiety, and I advise you to bear this carefully in mind. Such evidence of failure of the respiratory power may sometimes precede failure of the pulse, and is always of anxious significance. In addition to these serious warnings we observed that our patient tended to sink down in her bed, that she became mentally dull and listless, suggesting diminished excitability of the nerve centres, a conclusion which was further confirmed by the involuntary passage of urine and stools. These presages of imminent danger were only too fully realised, and the patient rapidly sank, and died five days after her admission to the hospital.

The post-mortem examination was in entire harmony with the diagnosis. No macroscopic change was found except evidence of extreme fatty degeneration of the heart,—doubtless the result of the extreme anæmia,—and the changes in the red bone marrow recognised as characteristic of pernicious anæmia. The marrow was soft and diffuent in consistence, and had a peculiar greyish-red appearance—features which are distinctive of the disease.

In reference to treatment little could be done. The vomiting and diarrhœa were, from shortly after her admission, such commanding symptoms that it was difficult to give even reasonable nourishment to the patient, not to speak of the administration of arsenic or other medicinal agents directed to the cure of the anæmia. Transfusion has been practised in such cases, and the late Dr. Brackenridge, of Edinburgh, published several cases in which he had obtained successful results. On the other hand, in advanced cases the practice has been known to precipitate a fatal issue, and I did not feel justified in advising it in the present case. The patient, indeed, when she came to the hospital was in so serious a condition that it was almost impossible to hope for any degree of improvement.

You have now before you all the facts of the case, and I have felt it advisable to review them at length, because for many reasons the case is one of great importance to you. In the first place, it introduces you to the manifestations of pernicious anæmia in

a fairly complete form, and may be usefully compared with the descriptions you find in your systematic text-books. It very distinctly illustrates how insidious is the approach of this disease, and how its course may be completely free from any striking or dramatic incidents until the fatal termination is actually in sight, and the chance of successful treatment is reduced to a minimum. This consideration will impress upon you the necessity of recognising the disease, when possible, in its early stage, for undoubtedly when recognised early it is susceptible of improvement, if not of actual cure; complete rest in bed, and the administration of arsenic for a prolonged period being, in my experience, the most reliable therapeutic measures to adopt. The safe position for the physician to hold is never to be satisfied, at least in an adult, and especially in a male adult, with a diagnosis of anæmia. No doubt in girls and young women we do often find the form of anæmia termed chlorosis, which, as far as our present knowledge goes, is a disease in itself, occurring without any recognisable primary cause, unassociated with facts carrying a serious prognosis, and readily susceptible of cure under the steady and prolonged administration of iron. But except under these conditions a diagnosis of anæmia is always an unsatisfactory one. The condition may be the direct result of loss of blood, and its position and treatment are then obvious. On the other hand, it may be the most conspicuous feature of such serious diseases as were considered in the differential diagnosis of the present case. It is therefore your duty to remember this, and by the appropriate methods of examination to fit yourself to advise and treat your patient. If you err in this respect you may readily allow him to proceed with a most imperfect perception of the gravity of his condition, and with most inadequate treatment, until by the progress of the disease his position has become practically hopeless. The case carries with it also some practical teaching as to the attitude of the physician towards cases in which fever without appreciable physical signs or localising symptoms, exists, and in all these respects it is worthy of a place in your note-books and your memories. It is a useful contribution to your medical education and to the most serviceable of all medical text-books, viz. the one that is written in your own experience.

ON THE TREATMENT OF INGUINAL HERNIA.

BY
W. ARBUTHNOT LANE, M.S.

GENTLEMEN,—If we hope to arrive at scientific methods of treatment of this condition we must consider the anatomy and physiology of the part, and the manner in which a hernia is produced.

By inguinal hernia is meant the escape from the abdominal cavity of some of its contents into or through the inguinal canal.

The arrangement of the inguinal canal is such that the cord passes out from the abdomen obliquely, its points of entry and exit being spoken of as the internal and external abdominal rings respectively. In the interval the cord lies between opposing layers of muscle and tendon, which constitute the abdominal wall.

It is the obliquity of this channel of exit of the cord from the abdomen that ensures the security of the wall. Even in infancy, when the peritoneal cavity is in direct communication with the tunica vaginalis, such sudden increase in intra-abdominal tension as occurs in the normal abdomen from crying, &c., acting at right angles upon the inner aspect of the abdominal wall, forces the planes of muscle into apposition, and prevents the intrusion of the viscera along the track of the cord. If, however, the intra-abdominal tension be increased very greatly, the muscles forming the wall are stretched and over-strained, and cease to perform their function normally.

Owing to a diminution of tonic contraction in the muscles, the alteration in the form of the abdomen consequent on the stretching of the several components of its wall, and the continued presence of an excessive intra-abdominal tension, the obliquity of the canal is diminished, and the contents of the abdomen are forced along it.

I would refer to a short paper on "The Causation and Treatment of Hydrocele and Hernia in Young Infants," which was communicated to the Section of Diseases of Children at the Annual Meeting of the British Medical Association held in Newcastle-on-Tyne in August 1893, since in it

was described, I believe for the first time, the manner in which hernia and hydrocele are produced in infancy by the increase in intra-abdominal tension produced by flatulence and indigestion, the irritation of the intestines causing a variable amount of peritoneal effusion; the latter percolating through the processus vaginalis produces the hydrocele of young life with which we are so familiar, and which the surgeon imagines he can treat by what he calls discutient lotions applied to the scrotum, and by aspirating the contents for the time being of the tunica vaginalis. Of course, this treatment is based on no principle whatever.

The presence of such fluid in the peritoneal cavity of young children suffering from hydrocele can be readily detected by placing the child in the erect posture, when it may be sufficiently abundant to give very deficient resonance, if not dulness, in the lower part of the abdomen. By carefully tapping with the finger or spatula a distinct thrill can usually be detected in the lower portion of the abdomen. In attempting to obtain a thrill through fluid you must remember that the facility with which this is detected varies inversely with the degree of tension of the fluid. This you can prove at once by means of one of the large hydroceles that abound in our out-patient rooms. Choose a loose one, and holding it lightly in the left hand, tap the mass lightly with a penholder. The thrill transmitted by the fluid is felt most distinctly by the hand grasping it. Constrict the upper limit of the hydrocele by means of a bandage, so as to gradually increase the tension in the sac, when you will find that the thrill becomes less and less obvious till it cannot be obtained at all; or you may demonstrate the same on a rubber bag under varying conditions of distension with water.

I mention this, as when showing this thrill in a subject standing erect, and in such a position that the abdominal wall is as lax as possible, I have been met by the statement that the thrill is not that of fluid, but is a movement transmitted along the abdominal wall, and this objection has been supported by the fact that a book or bed-card placed vertically and driven into the abdomen in the middle line reduces its distinctness. This it certainly does, and you will find that the loss of distinctness will vary directly with the increase in intra-abdominal tension which results from the pressure exerted. To put such a child

on its back and to attempt to obtain an indication of the presence of fluid in the abdomen by percussion or by a thrill will very rarely afford any result; since the amount of fluid is usually small, and in this posture is distributed over a very much larger area.

In some cases of hydrocele, when the processus vaginalis is large, the fluid in the hydrocele may be diminished gradually in quantity as the child is brought slowly from the vertical to the horizontal posture, and the reverse is true in bringing the child from the horizontal to the erect posture. As a matter of fact, I have verified these conditions on the operating table, and it was from such observations that I arrived at the conclusions contained in the paper just referred to.

It seems to me that in the examination of the abdomen, we are not sufficiently familiar with the advantages to be gained by examining patients in various positions, and that we confine ourselves almost entirely to the supine. For instance, I might mention that method suggested by one of our late surgical registrars, Dr. Alfred Parkin. He found out that in the easy sedentary position, the abdominal wall being thoroughly relaxed, owing to the descent of the anterior part of the liver for a considerable distance, it is possible to feel its edge, its under surface as far back as the transverse fissure, and any coarse lesion of the gall-bladder and bile-ducts, together with a larger area of the upper surface of the liver than in the recumbent posture.

It would be difficult to exaggerate the advantages which are sometimes gained by the use of this posture. But at the same time, while in a loose flaccid abdomen one's fingers quickly become thoroughly accustomed to the feel of the liver, gall-bladder, &c., in a fat tense abdomen one may spend a good deal of time in acquiring very little information. It is well to bear in mind that if you do not succeed in feeling as much as you would like on your first attempt, half an hour spent in familiarising yourself with the feel of the several parts will probably enable you to do so.

Returning to our subject. In infancy, as in adult life, the presence of a quantity of fluid in the tunica vaginalis assists in bringing about the escape of bowel and omentum from the abdomen by the strain exerted by its weight through the cord upon the outer limit of the posterior wall of

the canal, namely, upon the conjoined tendon, the obliquity of the canal upon which its security depends is lessened and the internal ring is approximated to and finally brought immediately behind the external abdominal ring. This is also increased by the pressure exerted by bowel or omentum in an existing hernia.

While the track is yet oblique, the pressure exerted by a truss on the inguinal canal forces its walls together, and, assisted by the intra-abdominal pressure, prevents their separation by bowel; a very moderate amount of pressure upon the outer wall of the canal seems to effect this, providing its posterior wall is normal.

When the internal ring is so placed as to lie immediately behind the external, the difficulty of retaining the intra-abdominal contents is tremendous, since it is only by the pressure of the pad upon a bung-hole orifice that they can be prevented from escaping. Movement of the pad in any direction freeing a portion of the aperture from considerable pressure allows of the escape of bowel, &c., from beneath the margin of the pad of the truss. The amount of pressure which is requisite to close this orifice is often so great as to become unendurable.

It also depreciates still further the mechanical disabilities of the patient, in that it increases the size of the aperture and so adds to the difficulty of preventing the escape of bowel or omentum. Between the extremes of a canal of almost normal obliquity and that in which all obliquity has been lost there is, of course, every intermediate variety. It is therefore necessary that our treatment of cases of inguinal hernia shall vary with the particular mechanical conditions present.

The general feeling of the present day in the face of the comparative safety of surgical interference, even when in the hands of an operator possessed of little skill and less cleanliness, has tempted surgeons to resort very readily to such active measures as are comprised under the comprehensive term "radical cure."

This may mean almost anything, and I will not attempt to define it, nor describe to you the innumerable methods and modifications of methods that have been devised to meet the difficulty.

Very often some endeavour is made to improve on nature, and almost always the operation is regarded as producing a mechanical condition of

such a character as to render it impossible for the contents of the abdomen to escape again in the same situation, although it continues to be exposed to the same factors that determine the formation of a hernia in the first instance.

I have read a great many accounts of the success of various operations, and of the ability of the patient to discard the use of a truss after operations. I have also performed for many years the duties of surgeon to the National Truss Society, and I have seen a large number of patients who have been operated on and in whom a hernia has again formed. In our technical language such are described as "relapses."

I have asked these patients why they had not returned to the surgeons who had operated on them, if only for the reason of helping them to correct their statistics, and have been invariably met by an answer to the effect that they did not intend to be cut about any more. Many asserted that the operation has not only not done them much good, but harm.

I am not venturing to criticise any particular operation, but it seems to me unreasonable to expect that however skilful the operator he can produce in the working man a mechanical condition better than the normal.

This canal was presumably normal before the hernia developed, and even if rendered as good as it was before it yielded, if exposed unaided to the same forces it is likely to give way again.

Certainly my experience of the patients of this Society impressed me with the idea that it is not wise to rely too much on statistics, however carefully they have been prepared, and however conscientious the compiler may be.

The principle that has guided me in operating on cases of inguinal hernia is to restore the mechanics of the canal as far as possible to the normal. To do this it is necessary to fully expose the cord by splitting up the outer boundary of the canal, namely, the aponeurosis of the external oblique along the length of its fibres. The cord is drawn outwards, and the conjoined tendon exposed and defined. Its free margin is stitched to Poupart's ligament so as to constitute again the posterior boundary of the canal. The neck of the sac is ligatured, and the sac removed. To utilise the sac as a plug is based upon the false assumption that this structure retains its character after

the factors which determined its form, &c., have ceased to exist. The incision in the external oblique is closed, and if necessary any slack taken in.

The patient is kept absolutely at rest in the recumbent posture for five or six weeks in order to allow the sewn parts to unite firmly, every precaution being taken to avoid the exercise of tension upon them.

All that I calculate on is that I put back the hands of the clock as regards the hernia, restoring the anatomy and mechanics of the part as far as possible to the normal; so that if the patient is of necessity exposed to the same forces that originally developed a hernia, he is ordered to wear a suitable truss as a precautionary measure.

We are sometimes inclined to forget that the human frame is only adapted to perform a combination of movements of activity and rest without undergoing change. Any deviation from this results, as I have shown, in a definite alteration of the anatomy of the part, whether it be the constant assumption of a resting posture resulting in its fixation and exaggeration, as in the feeble young subject, or the constant assumption of a posture or sequence of postures of activity resulting in their fixation and exaggeration, as in the powerful coal-heaver and coal-trimmer. In other words, the anatomy of a subject deviates from the normal in proportion as its physiology or mechanics are varied or specialised. In many occupations the abdominal wall, especially the inguinal canals, are exposed to a greater strain than they unaided are able to bear, and hernia is commonly produced. With such labourers it is wise to apply a suitable truss on the earliest indication of the intra-abdominal pressure being excessive.

In advanced life, when there is only a single aperture in the abdominal wall, the tissues are much stretched and wasted, and the hernia is large and a hydrocele possibly present, I prefer to remove the testis together with the sac, to sew up the aperture in the wall, and perform the only operation for inguinal hernia to which the term "radical" can be applied with any accuracy. I have found this procedure give the greatest satisfaction to the patient.

Returning to the infant and young child we recognise that the most important factor in the treatment of the case is to reduce the intra-abdominal tension to normal by feeding the child

at regular and suitable intervals. In many cases the hernia ceases to appear when this is effected.

If the child is old enough to wear a washable truss it should be worn constantly, and when removed for purposes of cleanliness the child should be kept on its back, and firm pressure exerted on the part in order to prevent the entry of bowel or even of peritoneal fluid into the sac.

The processus vaginalis being deprived of the pressure exerted upon it by the intrusion through it of bowel, omentum, or peritoneal fluid, undergoes its normal contraction and obliteration, which have been delayed by the presence of an abnormal force, and the hernia ceases to exist.

If, however, the posterior wall of the canal has been much displaced, and the abdominal wall is very lax, an operation similar to that in the adult must be performed.

In cases where there is no displacement of the posterior wall, but in which a truss for some reason cannot be worn, the canal may be split up, and a ligature put round the neck of the sac at the internal ring, telescoping in this way the more tedious and uncertain treatment by pressure. This applies chiefly to the treatment of the children of the poor, or of those who must enter a service, as the naval, at an early age.

There is a general consensus of opinion that while in the young subject it is possible to cure an inguinal hernia by the pressure of a truss, in the adult all that can be done by a truss is to prevent the entry of viscera into or their passage through the canal. So much so is this the case that the patient is directed to remove the truss on going to bed, and to replace it before assuming the erect posture.

As I have already told you, except in old-standing cases in which the aperture in the abdominal wall is a single one, the pad of the truss pressing backwards, inwards, and upwards, tends to increase or re-establish obliquity of the track occupied by the cord.

By means of a well-fitting truss worn continuously, it is often possible to prevent the entry of any structure into the neck of the hernial sac, and consequently to deprive its peritoneal surface of the pressure which determined its existence, and without the continued action of which it shrinks up and ceases to exist. It is upon the possibly unconscious knowledge of this, that many

irregular practitioners trade. They apply a truss that will effectually retain the contents of the abdomen, and they insist on the truss being worn continuously for a sufficiently long period of time to allow of this shrinkage taking place, because of loss of function. The difficulty the surgeon experiences is in getting the patient properly and accurately fitted with a truss, since the work is of the most difficult character, and has to be done by some one other than himself. I have known large hernial protrusions of considerable standing disappear, and the inguinal canal restored to its normal function, by this mode of treatment.

The case requires treatment other than local, and the special factors that determine the formation of the hernia must be recognised. For instance, cough, constipation, excessive fat, flatulence, stricture, piles, strain from occupation, &c., must, if present, be treated.

NOTES.

Pelvic Blood Collections and their Treatment by Vaginal Incisions.—Collections of blood in the pelvis, forming what is known as blood tumours were early recognised. Such a condition has been described by Ruysch in 1691. Such a collection was described by Recamier in 1831, in which the tumour formed behind the uterus following a miscarriage. Supposing it to be an abscess, it was opened, when congealed blood escaped. Cases were described by Velpeau, Bernutz, and others, but Nelaton in 1850 was the first to give a clear, intelligent account of the affection, which he denominated as retro-uterine hematocele. The term hematocele was at first limited to those cases in which blood was extravasated into the peritoneal cavity, but later was extended to collections of blood in the pelvis, either within or external to the peritoneum. Hæmorrhage most generally takes place into the peritoneal cavity, accumulating in its most dependent portion, Douglas's cul-de-sac, but it may take place also beneath the peritoneum, in the cellular tissue, in the broad ligament, in front of the uterus, or behind that organ. Intra-peritoneal hæmorrhage is far more dangerous, as where a vessel of considerable size ruptures there

is nothing to obstruct or prevent the bleeding, until the patient becomes so anæmic that a clot takes place into the vessel as a result of the weakened condition of the heart. The patient may die from shock or from profound loss of blood before this can take place. Hæmorrhage into the cellular tissue is much less dangerous, for the reason that it is to a degree self-limited. The accumulation of the blood by its pressure arrests the hæmorrhage. Internal hæmorrhage may arise from a variety of causes, each of which may produce either the intra- or the extra-peritoneal form. The most frequent cause is rupture of an ectopic gestation. Other causes are—escape of the foetal sac through the abdominal orifice of the tube, rupture of hæmorrhoidal vessels, tearing of the tube or pelvic adhesions by massage, examination of patient, or other traumatic lesions; rupture or perforation of the uterus; rupture or injury of an ovarian growth; twisting of the pedicle of an ovarian or broad ligament cyst; malignant disease of the ovary or tube; internal hæmorrhage during menstruation or following an abortion or miscarriage; slipping of the ligature following an operation, or a slipping back of the artery and hæmorrhage taking place into the broad ligament or stump.

The symptoms will depend largely upon the situation of the hæmorrhage. As has been stated, intra-peritoneal hæmorrhage is far more serious. It is usually attended with symptoms of shock, the patient being taken suddenly with profound anæmia, face blanched, pupils dilated, countenance anxious, sighing, irregular respiration, skin covered with cold, clammy perspiration, pulse feeble or entirely absent, heart beating rapidly and feeble. The patient may die within a few minutes as a result of the severe shock.

It was my unfortunate privilege to see a patient upon whom a diagnosis of ectopic gestation had been made, in which beneath the sac was a pulsation similar to that of the radial artery. Diagnosis having been made of ectopic gestation, the physician brought her with the privilege of making an examination, during which time I left the room. I was called back in less than ten minutes to find the patient presenting all the symptoms of immediate dissolution. The condition of rupture was recognised and an operation urged, but the consent of the patient and her daughter, who was

with her, could not be secured. Her husband was sent for, and arrived within less than an hour. The abdomen was opened, found to contain two quarts of fluid blood; the bleeding vessel was immediately secured and ligated, the abdominal cavity irrigated, but the patient only survived the operation a couple of hours; she did not rally from the shock.

Not all cases of intra-peritoneal hæmorrhage, however, result so fatally. In some the bleeding may be arrested early. In others it is slow, and the patient is enabled to partly maintain her strength in spite of the hæmorrhage. The symptoms of extra-peritoneal hæmorrhage are not so profound. The patient may suffer more pain from the tearing up of the cellular tissue, pressure upon the nerves, upon the bladder, or upon the rectum. There is a sensation of weight and pressure, of discomfort in the pelvis. Hæmorrhage may not be sufficient to produce marked shock, although the patient probably will present a history of having felt weak and faint. The extra-peritoneal hæmorrhage permits the recognition of a mass or tumour in the vicinity of the uterus. If in one broad ligament, the uterus may be pushed over to the other side of the pelvis, and the entire broad ligament or side of the pelvis filled up by the blood collection. Such a collection may press upon the bladder, giving rise to frequent desire to urinate, to sensation of vesical tenesmus. The pain, presence of tumour, more or less anæmic appearance of the patient, the history of disturbed menstruation or an injury, will generally be sufficient to permit of the diagnosis. Intra-pelvic hæmorrhage, however, does not afford any physical signs until after it has become encysted, when the history of a sudden onset, presence of a collection in Douglas's pouch, and the pulse of the patient should be sufficient to afford suspicion as to the character of the trouble. Where it has remained for a length of time it may become infected from the tube, or its proximity to the intestine, giving rise to septic phenomena. After the hæmorrhage has been arrested, unless the collection has become infected, nature shows a disposition to favour its absorption and disappearance. Such collections may be entirely absorbed after a length of time, leaving no, or but little indication of their previous existence.

In other cases they remain for a long period, give rise to secondary manifestations, possibly

suppuration, to opening by pressure into the rectum or vagina or bladder, and permitting the discharge of blood by one of these avenues. The most fortunate termination is the opening into the vagina. That into the rectum is usually situated so high up, not at a point at which the cavity can be thoroughly drained, but one which favours the escape into the sac of the contents of the bowel, with the increased danger of infection and suppuration. The treatment must necessarily vary greatly as to whether the collection is recent and still accumulating, or has existed for some time. It may be a question as to the wisdom of immediate resort to operation in cases of recent hæmorrhage. If we have reason to believe that hæmorrhage has ceased, and the patient has recovered from shock, certainly it would be unwise in such cases to advocate vaginal incision and the removal of the blood collection with possible displacement of the clot in the bleeding vessel, the re-development of hæmorrhage, with the inability to determine and secure the point from which the hæmorrhage has taken place. Certainly in such cases, if we have reasons to believe that hæmorrhage is still continued, the wisest plan of procedure would be to institute such measures as to promote the most favourable condition of the patient, and at once open the abdomen and secure the bleeding vessel, subsequently removing or not the blood collection as the condition of the patient may indicate.

The recognition of the fact that these collections of blood were in the majority of cases ultimately absorbed, has led many physicians to advocate a resort to the method of expectant treatment, in preference to operation. When we consider that these collections of blood are large, the possibility of their being infected by proximity to the intestine or a diseased tube, or uterus, the possibility of the clot becoming more or less partially organised, formation of thickening and adhesions, would certainly seem to make it advisable to resort to operative interference in preference to the expectant procedure. In all cases in which a collection has existed for such a length of time as to render probable the obstruction of the vessel and discontinuance of hæmorrhage, the collection should be evacuated, whether it be situated free in the abdominal cavity, encysted, or whether it may have taken place beneath the peritoneum, forming an extra-peritoneal blood collection.

The operative treatment, in such cases, through the vagina is preferable, for the reason that it permits of our reaching the blood collection and evacuating its contents with a far less severe and dangerous procedure than would be the opening of the abdominal wall and the bailing out of large collections of blood through the peritoneal cavity. Veit has asserted that the blood from an intra-peritoneal hemorrhage does not coagulate nor become encysted unless there has been previous inflammation of the pelvis and the formation of adhesions. This assertion, however, we cannot credit, as it has been our privilege to see quite a number of cases in which hæmorrhage has taken place into the peritoneal cavity, resulting in its being encysted in Douglas's pouch and presenting large masses of clotted blood. The vaginal operation should be a free incision and not a mere puncture, an incision which will permit of the introduction of two fingers and the pressing out and emptying of the clots from the pelvic cavity. The clearing out of the mass should be followed by irrigation with a normal salt solution, using a large quantity of this fluid, and at the same time manipulating the parts to remove and wash away portions of clot. While this is being done, the abdomen should be carefully held between the hands of an assistant, pressing its contents toward the pelvis. Incision preferably should be made by the thermo-cautery, as the cauterized surface will be less likely to close prematurely, and lock up the material in the pelvis. As the cavity is being cleaned, and being free from fibrin masses of clotted blood, the tube and ovary on either side should be carefully examined to ascertain their relation to, and responsibility for the hæmorrhage. If the tube presents evidence of having been ruptured, and contains a clot, it should be drawn into the vagina, ligated, and the clotted mass removed. The pelvic cavity should then be packed with sterilised or iodoform gauze, filling it up, which will favour drainage of serum and the escape of any morbid material still remaining. This operation, as we have said, should be a free vaginal incision; a mere puncture, or the use of the aspirator cannot be considered otherwise than reprehensible, as the puncture affords opportunity for introduction of infectious material, and does not afford a chance for subsequent drainage. We would then advocate vaginal incision and the

evacuation of blood collections, first, for the reason that in so doing we remove the possibility of its infection and subsequent suppuration; second, its removal leaves a clean surface which will subsequently resume its normal conditions and functions, while the retention of the clot could not but result in its partial organisation and the formation of unfortunate adhesions which cannot but influence unfavourably the future health of the individual; third, vaginal incision and drainage can be practised with but slight danger to the patient, far less, indeed, than would result from the retention of the blood collection.—EDWARD E. MONTGOMERY, M.D., *Annals of Gynecology and Pediatrics*, June, 1897.

Stenocardia (Angina Pectoris).—H. N. Heineman of New York writes on this topic in the 'Medical News' of January 9th, 1897. After discussing the ætiological and other factors in these cases he recalls the fact that a great many attacks are excited by the influence of cold. The avoidance of cold water, and even of tepid water, to any large surface of the body on account of the succeeding chill must be advised. Such patients must wash their bodies one part at a time, and have that part well dried and covered before proceeding to the next. Even the keeping of the hands well gloved, maintaining warm feet, protecting the mouth and nose from the sweeping cold wind or air, are precautions that will save the patient from many an attack. The regulation of the patient's life and habits comes under this category. Avoidance of mental worry and emotion, of over-exertion, and even needless exertion at times, is imperative. The patient must walk with deliberation, and the combination of walking and talking may be too great a strain for the heart until it is improved by general treatment. Tobacco should be absolutely interdicted, and all spirituous liquors as well. In persons who have always taken wine, a small quantity of a light Moselle or claret, preferably Moselle, may be taken diluted at dinner or, exceptionally, if the patient feels weak and in need of it. Over-indulgence in food must be guarded against. If the patient has the prodromal symptoms of an attack, the seeking of shelter, of a warm room, of warmth to the surface, or resting lying down, may ward off an attack.

The general treatment must have two objects

first, the improvement of the heart and blood-vessels; and second, the relief of engorgement in the internal viscera and capillaries.

For the improvement of the heart and blood-vessels, the use of potassium or sodium iodide in from three to five grain doses kept up for months is an old remedial agent of a certain, though not always satisfactory value. The treatment by saline baths and by the Schott method of exercises has a most potent effect in improving the condition of the cardiac muscle and vessels, and appears to have a direct effect in making the attacks less numerous and severe, and even in causing them to cease during a period of months or years.

The exercises with resistance, or Schott movements, should be carried out in the way indicated by the writer in a previous paper read before the general meeting of the New York Academy of Medicine. The movements are made in such a manner that the attendant can easily resist or hinder the movement, and the patient in seeking to complete the movement must overcome the resistance of the attendant. The movements must be made with especial care and caution in these cases, and the resistance at the outset must be at a minimum.

The artificial saline baths should contain from one to three per cent. of salt, and from one-fourth to one per cent. of chloride of calcium, and should gradually be strengthened by the addition of carbonic acid. By a process now in the last stages of completion, it will be possible to prepare the saline effervescent and saline effervescent flowing baths by means of a simple apparatus, so that the carbonic acid will be intimately mixed with the water.

For the relief of visceral and capillary engorgement we may resort to medicinal agents. Although the baths and exercises do this to a marked extent, the administration of a half-grain of calomel (well triturated) thrice weekly at bedtime, succeeded the next morning by a dose of Carlsbad sprudel salts, adds materially to the effect.

After the visceral engorgement has been much relieved, we can now use our nitrites, and in some cases nitro-glycerine; for until visceral congestion is relieved it is only playing "hide and seek" to try to relieve the capillaries, which are immediately clogged again by the nearest congested viscus. Having accomplished this, we now find the

cardiac tonics—sparteine, strophanthus, strychnine, valerian, and in suitable cases digitalis—of the greatest utility.

The general tendency to anæmia and defective oxygenation must never be lost sight of, and general tonics, including the use of oxygen gas, as recommended by A. H. Smith, will be of excellent service.

The treatment of the attack includes the use of nitrite of amyl in from three to five drop doses poured upon a handkerchief, or taken from a glass pearl in similar manner and inhaled.

The use of nitro-glycerine in $\frac{1}{100}$ -grain doses is of less rapid efficacy. It may be repeated once in three or four hours, or even longer intervals. In a severe attack it may be given at half-hour intervals, but should be given guardedly. In some patients the effect of nitro-glycerine is immediate, in others very slow. Hypodermic injections of morphine, with or without atropine, is often of great service.

The use of ether or chloroform, with or without valerian internally, relieves moderate spasm, and is somewhat stimulating. Local counter-irritation, by issue, sinapism, or blister over the præcordium, or the actual cautery over the præcordium, or applied over the hepatic region in some cases, and the application of electricity, may be resorted to. Sometimes the local application of the hot water bag is of service. Exercises given with extreme caution have in the writer's own hands sometimes given some relief to the acute symptoms.

To recapitulate, he wishes to emphasise that the treatment should not be haphazard, but systematic, if we would accomplish anything; and considering the suffering entailed by this disease, and the danger involved, such attention upon our part is essential and called for.

First, we must remove exciting causes of every kind, and next relieve the visceral engorgement that has long existed because the heart was too weak and the vessels not elastic enough to keep up proper venous circulation; and having gotten rid of the *vis a tergo*, we relieve the capillaries by appropriate remedies and coincidentally stimulate the heart, which can now act better because its capillary embargo has been raised. The success of the treatment depends upon the following out of the sequence just suggested. The comfort to be guaranteed to the patient, and the prolongation of life, are sufficient rewards for patient toil and scientific interest.—*Therapeutic Gazette*, May 15, 1897.

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TORTICOLLIS.

Being the Second Hunterian Oration delivered before
the Hunterian Society, on February 24th, 1897,

BY

VICTOR HORSLEY, F.R.S., F.R.C.S., &c.,

Surgeon, National Hospital for Paralysis and Epilepsy ;
Surgeon, University College Hospital.

By torticollis we understand literally a twisting of the neck, but in effect the whole of the discomfort of the condition is due to the aberrant position of the head, which is produced not merely by twisting of the neck, but also by rotation and flexion of the head on the spine.

Ordinarily there are two kinds of torticollis described, the clonic form and the tonic form, but this is only dealing with the symptoms. What we ought to do at the outset of this subject is to endeavour to divide the diseased conditions according to the part of the nervous condition attacked, and this should be the foundation of our diagnosis, and therefore the basis of successful treatment. At the present time the text-books are curiously silent on this all-important point, and the term torticollis has almost assumed the status of a disease itself, whereas, of course, it is only a leading symptom.

Hence to-day we must begin by considering what portions of the central nervous system are capable of evoking the movements in question. It is quite obvious that there are four such parts of the nervous system :

1. The cortex cerebri.
2. The corona radiata.
3. The cerebellum.
4. The spinal centres of origin of the spinal accessory, and the first four cervical nerves. The peripheral trunks of these nerves also, if excited, are clearly capable of producing the condition ; but whether this occurs or not in actual practice, we will examine into directly.

The parts of the central nervous system that have just been enumerated may be excited by—

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1. Direct excitation, *i. e.* by inflammations, growths, or poisons circulating in the blood.

2. Indirect excitation, caused by the afferent roots of the cervical nerves being involved in diseased conditions, and so setting up reflex disturbance and excitation of the centres.

I must next proceed to describe the different varieties of torticollis that may be met with, and then we shall be able to arrange these according to the part of the central nervous system which we believe to be at fault.

Owing to the obviousness of the distortion of the position of the head, these varieties can be most easily arranged according to the position assumed.

The possible positions of the head are as follows :

1. The neck flexed, the chin bent forward towards the sternum, and the spasms frequently clonic ; from the nodding movement thus produced, therefore called nutans.

2. Deviation of the head to the side, *i. e.* with the chin pointing straight towards the shoulder, and the vertical axis of the head parallel to the vertical axis of the body.

This is the position of the head which in the lower monkeys is easily producible by stimulating the cortex, just in front of the gyrus precentralis inferior, and close to or within which area there is also the representation of the movements of the eyes (inwards of eye of the same side, and outwards of the opposite eye)—this latter being the movement known as conjugate deviation of the head and eyes.

3. Turning of the head to one side, and the tilting of the occiput backwards, so as to prominently elevate the chin.

4. Lateral flexion of the neck with protrusion of the chin towards the opposite side from that flexed.

5. Tonic or clonic spasms drawing the head backwards.

The next point is for us to consider the muscles by which these movements are produced, and their respective actions according to the side of the body stimulated.

Fortunately, through the special courtesy of Dr. Risien Russell, who has recently investigated the share taken by the different muscles involved, I am able to discuss the subject before you fully, for although his paper has not yet been published in the 'British Medical Journal,' he has sent for publication therein an elaborate communication in analysis of the spasm. Such analysis in the human subject being extremely uncertain, Dr. Risien Russell carried out a series of experiments on the monkey, the muscles and movements in which are practically the same as in man, and he has obtained some very valuable information.

1. *Nutans*.—The anterior nodding movement of the head is plainly produced by the straight muscles on the front of the spine, *e.g.* longus colli and the sterno-mastoids in part.

2. *Conjugate deviation of the head*.—This movement is in reality a very complex one, and its analysis is rendered more difficult by the fact that it has been so far found impossible to determine the exact share of the different centres and muscles in the production of the condition. This much, however, is perfectly clear, that when this highly co-ordinated movement takes place, the result is a simple instance of the correlation of muscles on the opposite side of the body, just in the same way as in conjugate deviation of the eyes, and as Beever and myself have shown, it also occurs in the ordinary deviation of the tongue. Thus the sterno-mastoid of the side from which the head turns and the splenius and other muscles on the other side, act in conjunction to produce this one deformity. If the neck be examined during the spasm it is very striking to witness the contraction, say of the left sterno-mastoid and then that of the right splenius and complexus, trachelo-mastoid, &c., forming a thick hard mass under the fingers, at and just below the sub-occipital triangle.

3. This form is simply produced by the muscles on one side of the neck contracting and dragging down the head to the corresponding shoulder, so that of necessity the chin is projected towards the opposite side; the muscles taking part in this are those supplied by the first and second cervical nerves on the one side, *e.g.* the right, as shown by Dr. Risien Russell.

4. In this form the condition is much rarer; it can be evoked from the cortex cerebri in the bonnet key at a point nearer the marginal gyrus.

5. The fifth variety is very rare, but of which I have seen two good examples. The occiput is powerfully retracted on the spine, which at the same time is rather directed forwards, as compared with the position of the shoulders. This appears to be produced by the spasm of all the muscles on the dorsal surface of the vertebral column with the doubtful participation of the trapezius.

Dr. Risien Russell finds all the cervical roots are arranged in pairs, *i.e.* the first and second, third and fourth, and that the uppermost pair has most to do with the lateral movement of the head in torticollis. Further, that in the remaining cervical nerves there is a slight amount of movement produced by their excitation, that movement consisting in a drawing of the head and neck backwards, partly directly; but as we come down to the origin of the first dorsal nerve we find that these movements of the head and neck are becoming quite indirect, and really owing to the innervation of the latissimus dorsi, that muscle dragging down the scapula and the scapular attachment of the trapezius pulling on the head. Further, he has shown, as in previous researches, the importance of remembering the fact that each muscle is practically supplied by two roots. For full details, and as a necessary guide to operation, the facts detailed in Dr. Russell's paper must be referred to.

From a survey of the just-described varieties of torticollis it is plain that the cortex cerebri is for the first three. The fourth and fifth varieties may be derived from the spinal centres, or even, especially in the latter case, the cerebellum; but though these are possibilities, there is no real evidence to show that these lower centres can cause the continuous or clonic spasm without there being a lesion exaggerating the functional activity of the cortical and higher centres.

The next question which suggests itself is whether we can assist ourselves in endeavouring to localise the origin of the spasm by observing the actual character of the spasm itself, for it is now fairly recognised as a result of all the experimental work of the last ten years, obtained by exciting respectively the cortex cerebri and the cerebellum, that whereas excitation of the cortex cerebri results in a combination of tonic and clonic spasms, excitation of the lower centres, *i.e.* the cerebellum and spinal cord, most generally produces tonic contraction.

Taking this position then as correct, we must examine the character of the spasm from another point of view altogether, namely, as to whether it is caused by organic disease of the nerve centres or nerves, or whether it is only a functional spasm.

It seems to me that this question is relatively an easy one to decide. Functional spasm, meaning thereby one produced by no gross disease that can be detected, is, as far as I have seen, invariably accompanied by other well-known symptoms of neurosis; that is to say, the person has a history of neurotic trouble, and presents the characteristic features of neurasthenia, *e. g.* insomnia, nervous irritability, with some anæsthesia, and paræsthesia and exaggerated reflexes, &c.

Further, as to the character of the spasm helping us to distinguish this division of the subject, there is no doubt whatever that functional spasm is essentially a clonic one, and if, as we have reason to believe, neurasthenic disturbances are most marked from their effects on the cortex cerebri, it is only reasonable from what we have seen also above that the result should be clonus rather than tonus.

I now enter upon what I consider the most difficult question in discussing the ætiology of this very severe affection, and that is the problem as to how far it can be produced or is at all produced by mischief in the peripheral nervous system; and in the first place before taking up that question we must review the nerve-roots which supply the muscles that are capable of inducing the aberrant position of the head and neck in torticollis. Fortunately there are very few nerves, comparatively speaking, engaged in this question, for of the cranial nerves there is only one which participates, namely, the eleventh or spinal accessory, and this, of course, supplies the sterno-mastoid and the trapezius muscle. The muscles on the posterior aspect of the spine, namely, the complexus, splenius, rectus, capitis posticus, trachelo-mastoid, semi-spinalis, &c., are supplied by the posterior primary branches of the first five cervical nerves.

Each of the spinal nerve trunks where it leaves the intervertebral foramen, divides immediately into a posterior and anterior branch, sending the posterior branch backwards over the articulating processes of the neighbouring vertebræ, the nerve often running in a little groove in the bone, and then branching out to supply the muscles. This

fact of the general distribution is useful to remember in the operation described below.

I will now mention such cases as are undoubtedly connected with the involvement of the nerve roots, and which appear to lend colour to the view that in some cases their direct involvement is the cause of the trouble. Such instances are those of direct injury to the upper part of the neck, as I have seen in two well-marked cases of falls while hunting in gouty subjects, and where a distinct swelling or thickening might be felt for a very long time after the fall; the fall unquestionably involving the posterior primary branches of just those cervical nerves which innervate the muscles spasmodically affected.

The other cases usually placed in the same category are extremely important ones, and almost entirely seen in children with caries of the upper cervical vertebræ producing torticollis; this, however, is not a simple matter, and in fact the mobility of the cervical vertebræ renders it far more probable that the aberrant position of the head is due to a forced position assumed by the patient to relax the pressure on the inflamed joints, because caries usually affects in this region one side of the spine more than the other. When such cases are healed the ankyloses that give trouble are sometimes very remarkable, the torticollis deformity being of course permanent.

This view of torticollis in caries is probably correct, as evidenced by the fact that when the disease is limited to the bodies of the atlas and axis, as it sometimes though very rarely is, then there is no torticollis, although the spinal roots must also to a certain degree be inflamed.

The diagnosis of caries under these circumstances of so-called torticollis is always easy; I therefore need not occupy your time by dwelling on this subject.

Similarly I need not do more than, for the sake of completeness, mention those cases of hæmorrhage into or inflammation of the sterno-mastoid as seen in infants; for these only result in a deviation of the head of greater or less degree, plainly attributable to the swollen muscle, and therefore offering no trouble or difficulty in diagnosis.

Treatment.—Having completed an ætiological sketch of the factors of this condition, we must next endeavour to make use of the conclusions arrived at

for treatment. Treatment hitherto of torticollis has not been satisfactory, for reasons undoubtedly that as yet it has not been found possible to diagnose either the seat of mischief or the nature of the mischief, *i. e.* whether the cortex or the lower centres were the seat of the abnormal action, whether the indisposition of the same was due to inflammation or to forms of gross disease.

Under these circumstances it is not surprising that treatment has practically resolved itself into (*a*) that suitable for functional cases, (*b*) that calculated to remove organic disease or its effects.

(*a*) Functional or so-called hysterical cases are of course best treated with the Weir Mitchell treatment, and need not be further discussed.

(*b*) The removal of organic disease would mean the direct removal of the cause of excitation affecting the nerve centres.

I may first quote a case which I saw in consultation with Dr. Poore, in which there was a definite attempt to treat a central lesion, and which, at any rate, succeeded. The patient gave a history of syphilis, and suffered from spasmodic torticollis of so severe a character that the body rotated with the head. No relief had followed a previous tenotomy of the left sterno-mastoid. Close to the middle line, and very near the coronal suture on the left side, was a scar from a blow. This scar was over the posterior ends of the superior and middle frontal convolutions, *i. e.* the region specially associated with the function of rotating the head. The patient was put under the influence of mercury, and ultimately practically recovered. This improvement prevented the employment of another method of treatment, *viz.* trephining and the exploration of the parts of the meninges and brain beneath the skull, which measures had been in contemplation pending the effects of the administration of drugs. Although this is a solitary example, it must be taken as a probable instance of the success which we may occasionally arrive at by attempting to treat these cases locally.

Accessory treatment is directed to quiet the nervousness, and electric treatment has been applied (1) to the affected muscles in the hope of altering their condition of activity, or (2) to stimulating the antagonistic muscles so as to successfully oppose the pathological spasm of their opponent. Surgical treatment has been invoked with more or

less success, and so far invariably by simply paralysing the muscles involved, or if not paralysing them throwing them out of efficient action. The latter *modus operandi* involves the detachment of the sterno-mastoid or its division at its lower end, so that it should have no *point d'appui* from which to turn the head. This separation of the muscle has either been done subcutaneously or by an open incision, but under both conditions it seems to me that it is a barbarously empirical line of treatment, and ought not to be practised.

A commoner but still empirical method is the division of the nervous supply of the muscles affected. For a long time it has been the custom in adopting this procedure to divide the spinal accessory, and undoubtedly this should be done, and without troubling you with the details of the various ways in which the spinal accessory has been reached for this purpose, I will briefly describe what I think to be the method which best enables the surgeon to reach and remove the nerve.

A two-inch incision is made along the anterior border of the sterno-mastoid, with its centre opposite the angle of the jaw. The fascia of the sterno-mastoid is opened, and the edge of the muscle is drawn outwards, the head being suitably and slightly flexed to admit of this being done with freedom. It is often possible during this preparation of the internal aspect of the muscle to detect the point at which the nerve enters it. The nerve to reach the muscle passes either in front or behind the internal jugular vein at an angle of 45° to the axis of the vessel, and usually, as far as I have seen in operations, behind the vein. If it has not been seen entering the muscle, then it should be looked for without any further dissection, as Keen originally described in similar operations when looking for the facial nerve, *i. e.* it should be looked for by electrical excitation (preferably unipolar, the other electrode being fastened conveniently to some other part of the body); using a weak faradic current; the exact site of the nerve in the floor of the wound will then be made very evident by the powerful contraction of both the sterno-mastoid and the trapezius when the electrode is pressed on the right spot. All that is necessary, therefore, is to dry out the wound, and to apply the electrode vertically at successive spots down the length of the wound, and noting the point at which maximum

contractions are obtained ; a little dissection with the knife will then expose the nerve.

Nerve-stretching is practically useless, and certainly it has proved to be so in my hands, the spasm returning as soon as the nerve regains its function.

When the nerve is divided a large piece should be excised to prevent the opportunity of reunion. Occasionally this is sufficient alone, but in a severe case the synergic muscles, *e.g.* the complexus splenius trachelo-mastoid, &c., of the opposite side must be deprived of the nerve-supply. The operation for this was, I believe, first designed and performed by Professor Gardner, of Melbourne, and subsequently independently proposed by Dr. Keen in America, and later described by Noble Smith. This operation is tedious, but very satisfactory in its performance as follows :—An incision is made from the occipital protuberance outwards along the superior curved line, and then downwards along the back of the neck until the level of the sixth cervical vertebra is reached ; it is carried freely through the trapezius, the splenius, and also complexus down to the laminæ. In a line practically joining the articular processes, the posterior primary branches of the first four cervical nerves are then looked for and wholly removed ; they are readily found as follows :—the first, or suboccipital comes out, of course, through the suboccipital triangle together with the plexus of veins, the junction with the vertebral, is buried with these veins in a mass of fibrous fat, and gives branches to the muscles bordering the triangle. It is extremely essential to remove accurately the whole of the nerve, and as the oozing is considerable it is best at once to dissect the fascia from the muscles forming the triangular space, and remove *en bloc* the contents of the triangle, and the deep veins. There is free bleeding, which is stopped with Wells forceps in the usual way, and finally the space explored with the electrode and the current, to ensure the complete removal of the nerve and its branches. The posterior branch of the second cervical nerve from its large size will have been seen in the early stages of the operation and preserved. It is simply followed down to the dorsal surface of the intervertebral foramen and completely removed. The third and fourth will be found most easily just crossing the lower border of the articular process by testing with the current, and then wholly removed. The result is

complete rest given to this part of the neck, so far as the muscles named are concerned. Incidentally it may be mentioned that Professor Gardner has removed the posterior branches of the first three cervical nerves bilaterally in three cases, and has found that there has been no disadvantage to the patient so far as preserving the normal position of the head and the horizontal plane of the visual axes is concerned. I have performed this operation myself five times, and certainly with very gratifying results ; in only one did I consider that the result was not good ; the patient was highly neurotic (a gentleman referred to me by Dr. Hughlings Jackson), and the spasms were very violent. The result was incomplete, as much torticollis remained. I believe that I did not remove the branches supplying the trachelo-mastoid ; but his case introduced a question left to this stage of my communication, namely, as to whether other muscles are not involved in the spasmodic affection. I have no doubt myself that the scaleni are occasionally involved, and that they were so in this case, and contributed not a little to the clonic spasm of the neck. Then also Professor Gardner has observed contraction in the omo-hyoid, and in another case I found contraction of the levator anguli scapulæ, and this has also been believed to be felt by others.

The occurrence of spasms in such supplementary muscles must be carefully determined before operation is proposed, because otherwise disappointment may result by the patient feeling a spasmodic contraction of the neck.

There remains a small point in regard to treatment which must be considered whether the treatment is operative or otherwise.

It has been observed by many who have treated this condition that it practically always attacks people after middle life, and very often those in whom there is a strong gouty history or tendency. Anti-gout treatment has therefore been adopted in addition to the neurotic treatment previously spoken of ; but treatment by drugs is very unsatisfactory, and in general it may be said that if the central lesion cannot be detected and treated, the peripheral nerves must be divided by the Gardner-Keen operation, and a good result may be confidently expected.

A NOTE FROM THE CLINIC

OF

DR. BEEVOR AT THE NATIONAL HOSPITAL FOR THE PARALYSED AND EPILEPTIC,

May 11th, 1897.

Cortical Lesion ; Operation ; Recovery.

This patient, the subject of my remarks this afternoon, was sent by Dr. Brown of Ipswich to this hospital in March, 1896, with the following history:—One day in November, 1895, he felt queer, with a peculiar sensation in the right hand and in the first finger, and noticed his hand was shaking. The movement spread up the shoulder, down the right side of the trunk, and then to the foot on the same side; he fell down and lost consciousness. Fourteen days later he had a second attack similar to the first. After a fortnight he had another attack. The first time that the fits altered in character was in this third attack, where it began in the foot, the arm being affected after the leg, the face escaping. Then he had fairly frequent attacks until he came here, being admitted for the first time about one year ago. About the time of the second and third fits he noticed headache at the vertex, which became more or less continuous. Then vomiting occurred almost daily. On entering the hospital the three chief signs pointing to lesion of the cortex were noticed: first, generalised attacks of fits; secondly, headache and vomiting; and thirdly, optic neuritis in the left eye. Iodides and mercury were ordered, though there was no specific history. This patient was not given any bromide. It is a mistake, I think, to give bromide when a growth is suspected which may be due to specific disease, because the bromide may mask the symptoms, and prevent the determination as to whether the improvement is due to the subsidence of the growth from treatment. On admission there was an eruption on the arms, consisting of roughness of the skin with some spots which had been observed during six years off and on; this might be thought to indicate syphilis, and further there was some slight scarring. The vertical headache he had was very severe and on the left side chiefly, and there was tenderness on deep pressure over the vertex. There was also distinct

weakness of the right side, the movements of the shoulder particularly being weak, and to a less extent in the wrist also. There was some slight weakness to be noticed in opposing the right thumb and finger. In the leg there was distinct weakness, chiefly in the ankle, and also in the extensors of the toes. The flexors of the knee were also weak, and also the flexors of the hip. The weakness was therefore chiefly in the lower part of the leg, at the ankle, at the knee, and in the shoulder. The knee-jerks were increased on the right side, but there was no ankle-clonus. In walking the patient dragged the right foot. There was no loss of sensation; there was optic neuritis with swelling to about four dioptries in the left eye.

The patient stayed in this hospital under anti-syphilitic treatment, and went out on the 10th May, 1896, when he could walk fairly well, and though he had slight headache the vomiting had ceased. The neuritis was rather less; he could move the foot fairly well, so as to bend the ankle up to a right angle, and he could invert the right foot, but not as much as on the left side. The movements at the shoulder also recovered. He went on for a time without having any fits, but in June the fits returned, and since then he has had one a fortnight. During this time treatment consisted of pot. iod.

He was readmitted on January 11th of this year. He could walk fairly well except for limping, the grasp was weak on the right side, and there was weakness in the right shoulder; in the right leg there was some slight deficiency. The right disc was normal as before, and the left was highly coloured; there was not much change in the vessels, and no swelling in the disc to be measured. The tender spot on the skull was just above the parietal eminence. Dr. Stoddart had the opportunity of observing a fit; it began with twitchings in the right foot, which went up to the hip, to the right shoulder, and down the arm. Dr. Hughlings Jackson has stated that the rule is for the face to escape when the order or march of the twitching commences in the foot. If the fits affect the face first, the twitching will start at the end of the fingers and go up to the shoulder.

The patient, I may say, did not lose consciousness; but in the fits he had at home, or rather in some of them, he was unconscious.

On consideration of the above conditions, and observing that the fits came back again after the patient had been put on specific treatment, it was determined to operate, and Mr. Victor Horsley having seen the case and agreed, the operation was accordingly done on the 15th. It was performed in two stages; the dura mater and arachnoid being exposed by removing the bone in the first stage, and then in the second, a week afterwards, the dura mater was opened and the cortex was explored. At the second stage of the operation a growth was found in the cortex on the left side, and a piece of the cortex containing it was removed along the upper edge of the hemisphere at the anterior part of the fissure of Rolando—in other words, a part of the fissure of Rolando was removed, and part of the median surface of the left hemisphere. The length of the part removed was about two inches. After the operation the patient had weakness in the right arm and leg; he could extend the thumb and index finger, but not the wrist and fingers. He had some slight anæsthesia of the upper arm, and some loss of sense of position in the right foot and hand, and there was some slight anæsthesia in the foot and leg also. The right knee-jerk was increased, and there was right ankle-clonus. Following on this he must have had a hæmorrhage or effusion, which very often in these cases may be of cerebro-spinal fluid, and there was pressure at the seat of the operation; at any rate, there was pain and complete loss of power in the right arm and leg, but not in the face. Then two days later on examining the patient there was found considerable loss of sense of position in the whole of the right arm, and also in the right toes, but anæsthesia had to a large extent passed off. On the 26th of February he began to move the right hand. You will notice that the hand moved first, and he could extend the fingers and the thumb, but there was no movement at the elbow or shoulder; there was also no movement in the latissimus dorsi on voluntary effort. There was no movement at all in the right leg, either at the knees or toes. On the next day he again lost some power in the right hand. On March 2nd there was still complete loss of power in the right leg; he could move the right hip a little, slightly flexing it. On the 9th he began to move the right thumb and the first finger, but not the other fingers, and he could just flex the hip, so that he began to gradually im-

prove in the fore-arm, but was unable to move the right shoulder or elbow. He could not shrug the right shoulder, and had still some slight loss of sense of position in the upper limb with the exception of the finger and thumb; also there was loss of sense of position in the right leg. On March 15th he began to bend his right elbow; he went on improving from that, and gradually recovered a certain amount of power in the leg, especially in the hip, and on March 24th he could abduct and adduct the shoulder, but he could not shrug the right shoulder, and the last note taken a week ago shows that he had all the movements in the right upper limb with the exception of shrugging the shoulder; he could raise the arm up in the air, and he could advance it by the serratus magnus, but he could not shrug the right shoulder. In the right lower limb he could not flex the knee; the extension, however, of the knee and hip are good, though weaker than on the left side.

The lesion in the cortex was probably an infiltrating gumma, although there was no history of specific trouble. He can perform the fine movements of buttoning and unbuttoning, which are really very complicated; these he can do very well, which you can see on his taking off his shirt to show us the shoulder movements. There is slight wasting of the arm, probably due to disuse. On testing him this afternoon there is a slight power of shrugging the shoulder, but it was absent last week. The important point is that this movement of shrugging the shoulder has returned last, and though it is certainly now much weaker than on the other side, still the movement has come back. In regard to muscular sense, on testing him you see this has completely recovered, and the anæsthesia has completely passed off that was noted some time back. With regard to the lower limb, he has not the power to move the foot, and no power to move the toes or the ankle. He can extend the knee, but he has no power to flex it. He can flex the hip and extend it. In testing the flexion of the knee it is advantageous to make the patient stand, and get him to lift the heel off the ground. This man never had any weakness of the rectus abdominis, the muscle so instrumental in raising a person who is lying down in bed, into the sitting position. The knee-jerk is now very active on the right, but there is no ankle-clonus.

This case had certain general signs and certain local signs. The general signs were those of headache, optic neuritis (affecting one eye only, the eye on the same side of the lesion, by a considerable amount of neuritis, viz. four dioptries swelling), and daily vomiting at certain periods. These are the three cardinal signs of intra-cranial growth. You may have any of these three signs in anæmia, but still these three taken together are evidence strongly in favour of there being an intra-cranial growth. The local signs which the patient showed were these: epileptiform attacks, beginning locally, and affecting only one side of the body, followed also by weakness on that side. They were not all typical Jacksonian attacks, because he had loss of consciousness in the early ones. If fits occur they need not be true Jacksonian fits; and in cases of intra-cranial growth, if there is loss of consciousness, it does not preclude the attack being due to an intra-cranial growth. Most of the attacks that this patient had began in the foot with a feeling of tingling, and then they went up the leg and along the trunk to the shoulder, and then down to the hand, where they stopped. The tingling then was followed by distinct convulsive movements, and in many attacks the patient had no loss of consciousness, only experiencing a feeling of weakness.

The question arises now as to the diagnosis of the seat of this lesion, and what was the nature of it. Since the operation the patient has gradually recovered power, and has had no return of the attacks. What was the seat of the lesion? What was the nature of the lesion? The attacks began in the foot, mostly in the big toe, and that indicated that the disturbance began in the motor area of the cortex where the big toe is represented,—that is to say, at the upper end of the fissure of Rolando on the outer surface of the hemisphere; therefore the fact of the fits beginning at the great toe was strong evidence of there being a lesion in that part of the cortex. The attacks spread from the toe to the hip, then to the shoulder, and thence to the hand, where they stopped. In this prepared specimen of the left hemisphere of a brain I point out to you in their order from above down along the fissure of Rolando the motor centres for the big toe, the ankle, the knee, and the hip; below them is the arm centre arranged in the following order:—the shoulder, the elbow, the first finger and

thumb opposite the bend forwards in the fissure of Rolando; below that comes the face area. At the operation the part removed was along the edge of the hemisphere, being exactly the area for the representation of the big toe, ankle, and probably the small toes. To put the case very briefly, before the operation one was justified in saying that he had an intra-cranial tumour because of the general signs—headache, optic neuritis, and vomiting; and from the local signs the tumour was localised, and found in the cortex where the big toe is represented. The question of the subsequent paralysis is of interest. After the operation the man was paralysed in the right arm and leg, and it seems quite evident that the amount of cortex removed was not sufficient to cause all this paralysis, and that it was simply a temporary thing. In some cases by merely opening the membranes and exposing the brain you are apt to have temporary loss of power of the opposite side, it may be from interference with the vessels, but if you remove part of the cortex there is much more disturbance, and one expects to find that the amount of paralysis produced is much more widely spread than can be accounted for by the amount of brain removed. The paralysis in this patient has been recovered from, and the part of the limbs which recovered first was that which is represented in the cortex at a point farthest away from the lesion, that is his right thumb and finger, then the wrist, the elbow, and lastly the shoulder recovered. Besides the cortex on the external surface it was found by Schäfer and Horsley in 1884 that the marginal convolution (the convolution on the median surface) is also excitable, and that there are obtained certain movements by stimulation, and the movements which they obtained from before back were as follows: forearm; humerus and scapula; trunk muscles; pelvis muscles; lower limb. The part which I am interested in is that for the movements of the humerus and scapula. There was enough of the cortex on the outer surface removed to account for the paralysis of the leg, but not enough to account for the paralysis of the shoulder; and I believe the latter was due to injury to this part of the marginal convolution, because when the edge of the hemisphere was removed some of the marginal convolution was also necessarily removed. It is certain that you cannot get the movement of

shrugging the shoulders from exciting the cortex on the outer surface of the hemisphere.

This opens up an important point with regard to the return of movements in a hemiplegia which is due to lesions of the cortex. It is said (the theory was first enunciated by Sir William Broadbent) that in hemiplegia, where the shoulder usually recovers before the hand, the reason is because the shoulder is less highly specialised. The shoulder muscles are so used to act together that they may be thrown into action from either cortex, whereas the specialised movements of the hands can only be excited by the cortex on the opposite side; therefore in hemiplegia the shoulder recovers first because the cortex on the same side can move it. This case we are now considering is rather against that theory, because here is a man with paralysis of the limbs on one side in which recovery has first taken place not in the least specialised part, but in the most highly specialised part, *i.e.* the finger and thumb. I think return of power was due to recovery from a lesion caused by functional interference, or perhaps exudation, and not to taking on of action of the hemisphere of the same side as the paralysed limbs; anyhow, though it was merely a temporary thing, still he has not recovered in the shoulder completely yet, though it is better than it was. In this case the centre has been destroyed which supplies the movements of the leg and the movements of the shoulder, and they have not recovered.

The question arises as to the bilateral and the unilateral representation of movements in the cortex. I would say that all the movements of the upper limb are unilaterally represented, the trapezius and levator scapulæ as well as all the upper limb muscles; and the lower limb muscles too, are unilaterally represented,—that is to say, only the cortex of the opposite side can throw them into action. It seems to me that in a lesion of the cortex affecting the leg centre, as in this case, the whole of the opposite side is paralysed at first owing to vascular disturbance. The parts recover with the exception of those whose seat of representation in the cortex has been removed, which do not recover unless they be bilaterally represented in each hemisphere.

In this patient the symptoms of a lesion of the cortex were fits beginning locally, followed by weakness and certain disturbances of sensation,

and the indications afforded by these disturbances are important. In the cortex the disturbance, which is produced after these fits, causes a certain amount of loss of sensation; but if you go still further and actually destroy the cortex the symptoms are paralysis of motion and sensation, a paralysis which is more or less permanent, and corresponds to the part of the cortex which is damaged. In this case the man has had a lesion of the centre for the leg, and especially of the part representing the big toe, paralysis of all the toes, and also of the ankle, and to a certain extent of the knee. But if you have destruction of the cortex you have also sensory disturbances as follows:—loss of appreciation of slight tactile impressions; inability to localise stronger tactile impressions though they can feel them; and inability to tell the position in which the different joints are placed. These symptoms were all marked in this man, but they have now passed off, the sensory before the motor paralysis. It is difficult to understand the small amount of loss of sensation which these cases often show. As to whether this man will recover power in the leg, I think is very doubtful. During the last week he has recovered to a certain extent the use of his shoulder, that is, he has taken three months to recover the power of shrugging the shoulder slightly, but in the leg there is very little recovery, and one can hardly expect much, considering the amount of cortex removed in the operation.

How is it, then, that his shoulder has so far recovered? One does not know exactly how much of the marginal convolution was removed, but probably there has not been sufficiently removed of the particular portion which supplies the shoulder to cause complete paralysis. The disturbance of the tissues may have been sufficient to cause complete paralysis for a time, but now he has after three months gained back some power in the shoulder.

With regard to treatment of intracranial growths, it is best to treat them with antispecific methods for six to eight weeks, and, if no improvement ensues, to recommend a surgical exploration if the position of the growth can be localised as in the above case.

DEMONSTRATION OF CASES AT CHARING CROSS HOSPITAL.

BY
DR. MONTAGUE MURRAY.

Exophthalmic Goitre.

GENTLEMEN,—This young woman presents the typical symptoms of exophthalmic goitre. The disorder began with an excited action of the heart. The palpitation was felt for more than a year before any other change was observed. Then the exophthalmos and the enlarged thyroid were noticed concurrently. She also presents Graefe's sign—that is, a delay in the drop of the upper eyelid when she looks down quickly. Stellwag's sign, retraction of the upper lid and widening of the palpebral fissure, so that the sclerotic can be seen all round the iris, is in this case sometimes, but *not always*, present—a fact which suggests that the retraction is due to spasm. The tremor is exceedingly typical—a fine tremor, about eight to the second. She is, moreover, very short of breath. No mental or emotional change can be made out. The pathology of the condition is very interesting. In its vascular phenomena it is almost the antithesis of myxœdema, the symptoms corresponding in effect to those due to an overdose of thyroid extract. But in two particulars the disease exactly resembles artificial myxœdema—the condition produced by the removal of the thyroid in animals,—there is *tremor* and there is *dyspnœa*. The tremor is of the same rhythm in both conditions. It was at first supposed that the dyspnœa depended upon pressure on the trachea, but it has since been shown that the dyspnœa was not lessened by removal of the supposed source of pressure. This was demonstrated by Horsley. At the same time he pointed out that exophthalmic goitre should be regarded as the product of a perverted action of the thyroid, whereas myxœdema is due to the entire absence of its action. Therefore one is not surprised to hear that when an attempt is made to treat exophthalmic goitre by thyroid extract, the effect is not so satisfactory as in myxœdema. When we are able to separate all the chemical products of the thyroid gland from one another, we shall doubtless find out which of

them will correct the vascular changes produced in exophthalmic goitre, and shall be able to cure exophthalmic goitre as readily as we can now cure myxœdema. The patient is anæmic, but not sufficiently so to account for the dyspnœa. The administration of digitalis, strychnine, and iron produced but little effect. She is now improving under belladonna. Phosphate of soda has been much recommended of late, and if necessary I shall prescribe it in this case. I believe the best thing is rest in bed, but she will not come into the hospital.

Cerebral Tumour.

This woman, who is 42 years of age, has had fits for five years. At first, these recurred every fortnight, then at longer intervals, until as much as a year intervened. The last few months they have become almost as frequent as at first. Since admission, one has been observed. The first movement noticed was of the right elbow, as if the patient were beckoning. Movements of the rest of the arm and in the corresponding side of the face quickly followed, and then the fit became general. In its other features it exactly resembled an ordinary epileptic seizure. The fit lasted two minutes. Drowsiness for an hour followed. No local weakness in the parts first convulsed was made out.

But to return to the history of the case. For the past twelve months the patient has been suffering from headache—mild and only occasional—and vomiting. This latter symptom has occurred nearly every other day, and quite independently of meals. Six weeks ago her sight began to fail, and she is now quite blind. If you examine her optic discs you will find a moderate degree of subsiding neuritis. Speech is normal, and there is no giddiness. There is some tenderness on the left side of the scalp, corresponding to the uppermost part of the fissure of Rolando.

In the absence of Bright's disease, vomiting, headache, and optic neuritis point to cerebral tumour. The history of the vomiting is not that of gastric or renal disorder, and there are no degenerative changes in the retina, such as one might expect in the case of kidney diseases. The scalp tenderness suggests the upper end of the fissure of Rolando as the seat of the growth, and the phenomena of the observed fit are consistent

with this indication. The representation of the shoulder and elbow movements are just below this area, but the part indicated by the commencement of the fit is not infrequently outside the place where the bulk of the tumour will be found. It will be advisable, however, to obtain a record of more than one fit before operative measures are considered; for we do not yet know if the fits always begin in the same way, nor can we yet be sure that no local weakness of the convulsed parts persists for a short time after the convulsions themselves have ceased. Whatever the nature of the tumour may be, it is slow in its growth.

The majority of tumours, because of their infiltrating character, or for other reasons, are not suitable for operation, and, in the case of those deemed suitable, the results are not always such as one perhaps feels justified in expecting. Moreover, the patient is not seriously inconvenienced by any of the symptoms, except the loss of sight, which no operation can possibly remove. It will be wise, therefore, to make further observations and to await the progress of the symptoms before resorting to surgery.

Some Results of Alcoholism.

This patient, a woman aged 36, was admitted a few days ago for "inability to walk."

Fifteen months ago she had an attack of retching and vomiting, in which the former predominated, and since that time has had, off and on, many similar attacks. About a month ago she complained of numbness below the knees, and of inability to walk or stand.

You will now see that when she attempts to grip your hand, or to raise her legs, the force exerted is very small, and that it is jerky and not continuous. You will also observe that she shows quite as much power in flexing her ankles as in extending them. Moreover, if we support her on both sides, and thus enable her to stand, she can raise her toes off the ground. When she attempts to stand or walk she keeps her feet close together, making no attempt to widen the base of support. The muscles of her calves are very flabby, and sometimes seem to be a little tender. There is distinctly defective sensation to touch and pain over the lower two-thirds of both legs, but this is much less marked in one great toe than elsewhere, and the upper level of the anæsthetic area varies from

day to day. Sensation to temperature is normal. The skin is very smooth and thin. The knee-jerks are slight but distinct. The plantar reflex is also slight, but a good deal of contraction takes place when it is being tested, giving the impression of voluntary control on the part of the patient. The only electrical change is a qualitative one—A.C.C. being slightly more marked than K.C.C. Her liver is uniformly enlarged, and the edge is not over-sharp.

An important point in this case is to determine how far the condition is due to multiple neuritis, and how far to functional disease. There is some evidence of the former in the history of gastric catarrh, the flabbiness of the calf muscles, and the slight electric change.

That the condition is not wholly due to neuritis is, I think, shown by the power retained over the flexors, the distribution of the sensory disturbances, the maintenance of the knee-jerks, the character of the attempt to stand, and the result of testing for plantar reflex. Alcoholism is doubtless the principal cause of the functional change, as well as of the neuritis.

Cirrhosis of the Liver.

The next case is a typical instance of cirrhosis of the liver. I am showing it because it illustrates two points that have recently been discussed with regard to the etiology of the disease. You will note that the liver and spleen are both large, and that there is marked ascites. The man's age is 42. When he was twenty he began to drink fairly freely, and continued to do so until he was thirty-two years of age. He, however, says that he never went much beyond four pints of beer a day. Well, one frequently encounters people who can stand that amount of beer without showing any symptoms of cirrhosis. After the age of thirty-two this patient knocked off a considerable amount of the four pints, and he had no symptoms until two and a half years ago. Then he had an attack of hæmatemesis. That passed off; he was warned, and became still more careful about his drink. He had no return of hæmatemesis until last Christmas. His third attack was last month, and he has had a fourth attack to-day. The chief point of interest is that he had no gastric catarrh until last October. I think we were all taught that spirit-drinking was very much more likely to

produce cirrhosis than beer-drinking, and when a thing is stated with great emphasis one believes it. Considerable evidence has been recently adduced to show that this statement is wrong, and that beer is quite as likely to produce the disease as spirits. But all this is "history," and open to many fallacies. It used to be taught that the concentrated alcohol circulated through the portal system and irritated the tissues lying round the vessels, and that thus chronic inflammation was set up. But quite apart from the fact that beer-drinking has been observed to cause cirrhosis as frequently as spirits, there have been some experimental researches, and cirrhosis has been produced in animals. So long as the experiments were confined to the administration of alcohol they failed. When, however, certain micro-organisms were used with the alcohol to produce catarrh of the stomach, cirrhosis of the liver began to appear. The etiology of this disease has, therefore, been stated to be as follows:—Catarrh of the stomach is produced; certain micro-organisms are thereby enabled to thrive. The toxins produced by these are carried to the liver, and there their action gives rise to the cirrhosis; so cirrhosis is, more or less, a germ disease after all—like everything else. Here, however, is a man who had hæmatemesis two years before he had any gastric catarrh. One case does not prove much, and "histories" are, as I have said, unreliable; but still I have questioned the man very closely, and he holds to all his statements. At any rate, the possible causal relationship makes it all the more necessary to treat the catarrh of the stomach carefully, quite apart from ceasing the intake of alcohol. This patient is having Carlsbad salts and iron.

Scurvy.

The next patient is a man æt. 45. He came in three weeks ago for "swelling in his right calf." It was then much larger than it is now. It was also painful, and the skin over it was discoloured, as if bruised. His left thigh was similarly discoloured. His gums were spongy, his teeth filthy, and his breath foetid. On inquiry, we found that his diet had been very restricted in amount, and had been limited to bread, butter, bacon, and blunders. The probability was that he was suffering from scurvy. The prominent fact in scurvy is hæmorrhage, the ubiquity of which furnishes nearly

all the symptoms of the disease. The small petechiæ round the hair-bulbs of the skin are the slightest forms which the hæmorrhages assume. Then there may be hæmorrhages into and from the mucous membranes. The gums are spongy because hæmorrhage takes place into their substance and they bleed; moreover, the conditions under which scurvy arises are those in which the mouth is altogether neglected, and decomposition is likely to occur. Decomposition acting upon gums which are damaged is more likely to cause ulceration than when acting upon ordinary healthy gums. It is also known that this condition of gum is only typically developed where there are teeth. If a toothless man gets scurvy he will have but little sponginess and no ulceration. This is because decomposing material tends to collect in the neighbourhood of the teeth. Bleeding also occurs in the alimentary mucous membrane, as well as in the nasal mucous membrane, and hæmatemesis, melæna, and epistaxis result. It takes place, also, into the muscular tissues, into the subperiosteal tissues, and into the synovial membranes, and therefore into the joints. This man has had no effusion of blood under the periosteum or into the joints.

Night-blindness is one of the classical symptoms of scurvy. In an examination paper a student puts it first. This man has not got it, and I have never seen a patient who had it. Scurvy may have to be diagnosed from pernicious anæmia, but it could hardly be mistaken for it unless the hæmorrhage is confined to the retinal arteries. Hæmophilia and purpura might give rise to the bleeding in the gums. From the latter, scurvy is distinguishable because the hæmorrhages are not so uniform in size, and you nearly always get them into one of the other tissues I have pointed out. The history will suffice to dispose of the former. There are cases in which the scurvy has been overlooked when the hæmorrhage was subperiosteal, and the man was believed to be suffering from syphilitic nodes. It has also been mistaken for rheumatism, because hæmorrhage had taken place into the joints.

People talk about scurvy as if they knew all about it, probably because it is known what deficiencies of diet produce scurvy, and what additions to diet will therefore suffice to cure it, namely, fresh vegetables. Beyond that, however, we cannot go. You are aware there is one theory

attributing it to diminished alkalinity of the blood, and another to a deficiency in the potassium salts. Possibly we shall find that defective coagulability is the chief cause of the hæmorrhage, and that this is due, as in some other diseases, to a diminished amount of lime in the blood. Hæmophilia, purpura, chilblains, urticaria, in which there are effusions from the vessels, are all treated very successfully by the administration of drugs which increase the coagulability of the blood. Professor Wright, of Netley, published some cases in which he showed that in urticaria the coagulability of the blood was diminished. He withdrew a small quantity of blood and tested it. Sometimes he found that the process of coagulation took three times as long as it ought to take. Dr. Wright suggested that in these cases chloride of calcium should be given, because it is known that lime and carbonic acid increase the coagulability of the blood more than anything else. Chloride of calcium is best given in the form of varnished pills. In ordinary cases of scurvy the conditions under which it has arisen are so marked that we have only to reverse these and diet the patient properly, keeping him at rest, and giving him such things as lime-juice, lemon-juice, potatoes, or oranges. Lime-juice is more portable than potatoes, although there is reason to believe that potatoes will pull a man through as certainly as lime-juice.

Imperfect Treatment of Syphilis.

In these adjoining beds are two men, both about the same age; they were both in the army at the same time, both had syphilis ten years ago, and were both treated for it for only three weeks. Now one of them has aneurysm of the arch of his aorta, and the other has tabes. In the patient suffering from aneurysm there is a projection over the second and third interspaces, mainly on the right side. It is in that part that the aorta comes nearest to the surface. The swelling and pulsation are both very evident. The right pupil is rather larger than the left, and there is a good deal of stridor with a little tracheal tugging. In testing for tracheal tugging, take hold of the larynx and hold it firmly, avoiding as far as possible the pulsation from the carotids at the sides; you will then feel a slight tug down at each beat. There is a distinct difference between the radial pulses. Though there is marked stridor

there is no paralysis of either vocal cord. The stridor is due either to the direct pressure of the aneurysm, or to syphilitic disease of the trachea, most probably to the former. The patient had no symptom at all until a year ago, and then he went to a doctor because he had continuous pain in his chest, and cough. It is to the credit of the practitioner that on the very first occasion he was consulted the aneurysm was diagnosed. Our patient was warned and treated accordingly. Continuous pain in a man's chest should always be considered seriously. This was one of the first clinical points I learned at the bedside. A man does not get continuous pain in his chest from a slight or functional cause. He is having iodide and a full diet. I once heard the late Dr. Bristowe call attention to the fact that the only thoroughly cured aortic aneurysms are those found for the first time on the post-mortem table, and he added that perhaps the best thing for a man with aortic aneurysm is to let him go about his work. But the answer to this is that those aneurysms which are not cured by this treatment are precisely those we are called upon to deal with.

The patient in the next bed, whom I mentioned in connection with this one, has a perforating ulcer in the front part of the sole. A year ago it began as a corn, which was cut, damaged, and then never healed.

This is the usual history of these perforating ulcers. The bursal cavities become septic, and the septic material penetrates into the deeper parts. The first thing the man complained of was inco-ordination—a year ago—followed by pains in the legs and stomach. His tactile sensation is blurred. He has no knee-jerks, but his pupils react to both light and accommodation.

Epidemic Cerebro-spinal Meningitis due to *Meningococcus Intracellularis*.—Stoeltzner

has reported a further case of cerebro-spinal meningitis in which the meningococcus intracellularis was isolated from the spinal fluid obtained by lumbar puncture. A child, 2½ years old, was seized with symptoms that rendered the diagnosis of cerebro-spinal meningitis probable. During the progress of the attack lumbar puncture was practised, evacuating turbid fluid, in which were found pus corpuscles and meningococci intracellularis.—*Berliner klinische Wochenschrift*, April 19th, 1897, p. 333.

A CLINICAL LECTURE ON A CASE OF SUPPURATIVE PERITONITIS.

BY

EDMUND OWEN, F.R.C.S.,

Lecturer on Clinical Surgery at St. Mary's Hospital.

GENTLEMEN,—This little boy, Charlie, was admitted on Thursday evening, March 14th, looking extremely ill. His belly was swollen, fixed, and tender, and he had been vomiting. Mr. Burrow, the house surgeon, recognised the serious condition of affairs, and immediately sent for me.

I arrived within an hour of the boy's admission into hospital, and found him in a state verging on collapse. His face was pale and drawn, his pulse was 75 and "wiry," and his temperature 100·8°. His lips were dry and covered with crusts, and he complained of great thirst, and also of being cold.

The abdomen, as I say, was distended and scarcely moving with respiration, and though tender all over, was especially so in the region of the right iliac fossa.

All this is duly recorded by Mr. Gribbell, the dresser who has had charge of the case.

In addition to these symptoms, there was a tender red swelling at the navel of about the size of half a Tangerine orange, which looked as if it might be caused by the presence of the inflamed sac of an umbilical hernia.

The history which his father, a butcher's assistant, gave was that he had always been a healthy boy, and that he had passed the sixth standard of the Board School, which shows that he was also a bright boy; that on the previous Saturday he had complained of pain on the left side of his belly, which was taken to be an ordinary "stomach ache." But as he was complaining of it again next day, the father went to a dispensary and got him a couple of pills. These did no good, and on the Monday he sent for the dispensary doctor, who treated him *secundum artem*, until Thursday—the day of his admission—when he called another doctor into consultation, and they agreed that the only chance for the boy was to send him into hospital for immediate operation.

Having examined the boy, I came to the con-

clusion that he had acute peritonitis, but whether it was caused by a strangulated umbilical hernia, which had possibly ruptured, or by perforation of an inflamed vermiform process, or by something else, it was impossible to say. Inflammation of the appendix is, of course, the most likely cause of suppurative peritonitis in a child, but in this case, though there was great tenderness over the right iliac fossa, the boy's first complaints had been on the other side of the belly. Then, if the trouble were originally appendicular, what was the nature of the inflamed swelling at the navel? The case, however, was one for prompt operation rather than for speculative and clever diagnosis. In many a so-called "belly case," the diagnosis can be made only when the peritoneal cavity is opened: previous to this everything may be obscure and uncertain. And in many a case it is well that the patient or his friends should be made clearly to understand that to open the belly is not to be dreaded as a terrible operation, but as a welcome and efficient method of finding out exactly what is wrong, and of then endeavouring to put it right.

Having gone thus far in the investigation of Charlie's case, Mr. Burrow and I called up the father, and I explained that something had gone wrong in the boy's belly, that no one could say exactly what had happened, but that it was very serious, and that I must make an opening for inspection, and, if possible, put it right; but that even if I did put it right I could not promise much, as the outlook was very dark, but that an operation was the only thing that could possibly save the boy.

The father was unusually calm and reasonable, and seemed, by his remarks, to take a very intelligent view of the case. When I had finished my little address to him, he said, "I leave him in your hands, sir; do what you think best. I am a butcher myself."

Whilst I was talking to the father the boy was lying on a hot-water mattress upon the operating table, and Mr. Gribbell and his colleagues were washing and preparing the surface of the abdomen for operation.

Having got the boy under chloroform, I made an incision downwards in the median line, beginning just below the umbilicus, so that I might first see what was the nature of the inflamed

swelling. There was, however, no communication discoverable between the general peritoneal cavity and the umbilical tumour, so I passed my finger down towards the region of the cæcum, and, as I was doing so, some stinking serum and pus escaped through the wound. Passing in two fingers, I then felt for the vermiform process; it was swollen and hard, and I could detect a concretion in it. I then made a second incision directly over the vermiform appendix, and let out a lot more foul fluid with fæcal odour.

The appendix came at once into view; it was free in the peritoneal cavity, and occupied a position external to the cæcum. There was a concretion at its end; but what was worse, there were two other concretions at the root of the process, which had set up a perforating inflammation just where the process came off from the cæcum. The inflammation, moreover, had extensively implicated the adjacent part of the cæcum, the oedematous and rotten wall of which was traversed by the same ulcer that had perforated the root of the appendix.

To remove the appendix was a simple matter, but what was to be done with the opening into the bowel? To turn in the edges and use Lembert's suturing was out of the question, and to perform a resection or any plastic operation would have been to run the risk of not getting the boy off the table alive. So I merely clamped the opening with three pairs of catch-forceps, and left their handles sticking out of the iliac wound, in order that they might insure the free escape of subsequent discharge.

The intestinal opening being thus temporarily blocked, I washed out the peritoneal cavity with abundant saline solution. And here I would remark that when, at an operation, I call for hot lotion for irrigation, I take good care that I get it. My experience is that, notwithstanding all the good qualities of dressers and nurses, they do not always seem to know that a warm lotion is not the same thing as a *hot* lotion. The result is that when one is washing out a large suppurating cavity, the irrigation fluid, by the time that it has got out of the reservoir, through the tube, and over the tissues, is barely warm, or is actually chilling. Such a fluid is of little value in the suppurating peritoneal cavity of a boy in a state of collapse, whilst a really hot saline solution is not only a

cleanser, but it is a valuable stimulant, and thus greatly helps in diminishing the shock of the operation, and tiding over the crisis.

In order to secure efficient irrigation in this case, I drew most of the intestines out of the median wound and washed them over with abundant saline solution, which dressers and nurses, in their determination to place themselves beyond risk of reproach, had made so hot that it was as much as I could do to bear my hand in it as I washed out the pelvic cavity, the splenic and hepatic recesses, and the crevices along the colon. I used no sponge, but with my hand in the belly, I gave every part as rapid and thorough a sluicing and cleansing as it was possible to obtain. Then I closed the median wound, but left the iliac one open, so that whatever would might find free escape.

I also passed a large drainage tube by the side of the catch-forceps down into the appendicular region. Then we placed absorbent dressings over all, and quickly got the boy into a bed which had been duly prepared and warmed for him.

During the night he had injections of strychnine and morphia, and he was allowed sips of hot water. Next day he looked better, though he was, of course, extremely ill, his pulse running at 130 per minute.

On the second day after the operation a little fæcal discharge took place alongside of the forceps, but the boy was looking better. The skin in the neighbourhood of the wounds was red and unhealthy; the suture punctures were septic, and some superficial sloughing was threatening. The abdominal wall was still hard, and there was but slight movement with respiration. There was tympanites, and the boy complained of pains and sickness.

In these circumstances I ordered him an enema of turpentine. Even at the risk of disturbing the inflamed track, it is often a good plan in these cases to get the bowel cleared of its stagnant fluids and decomposing solids. There are two ways of doing this, either by the administration of salts by the mouth, or, as in this case, by an enema. If a patient is very ill, and a speedy relief is desired, an enema is the preferable method.

The physiology of the treatment is that it enables the patient to get rid of products of

decomposition, which, if absorbed, give rise to a train of symptoms which, together, make up the clinical history of peritonitis. So far as I am aware, no surgeon has done so much in establishing the principles of this method of treatment as Mr. Lawson Tait, of Birmingham.

Mr. Gribbell says, in his notes, that the effect of the turpentine enema in relieving the boy's discomfort was marked, and that the general condition was improved. This was on the Sunday—the third day after the operation. The pulse was then 120, and the temperature about 100° F. But the temperature in peritonitis is not always a valuable indication, as, under depressing influence of ptomaine absorption, it may keep low, though all the time the patient is very ill.

On the fifth day, as the wound seemed likely to remain wide open, we removed the drainage-tube, and took off the catch-forceps.

There had been during the last day or two some faecal discharge along with the pus through this opening, which suggested that some sloughing had probably taken place where the forceps were nipping the edges of the wound. These forceps worried the boy, and as they were evidently no longer acting in a trustworthy manner, we had little hesitation in taking them off.

Day by day the amount of pus, flatus, and faeces passing through the wound diminished, and in due course the wound completely closed.

From time to time some of you saw the boy with me; you will remember the erysipelatous condition in the neighbourhood of the wounds, and how I explained that I by no means held myself responsible for it, as the incisions, and the sutures, the boy, and our fingers were all septic at the same time at our operation. The wounds, and everything about them, were teeming with the septic micro-organisms which had welled up from the peritoneum—bacteria coli, staphylococci, and germs of pretty nearly every sort. But, strange to say, though the skin about both wounds sloughed, where the peritoneal edges were brought together—as in the median wound and in the lower part of the iliac wound—primary union took place.

The lesson to be drawn from this is that the peritoneum—notwithstanding all the unkind things which have been said against it in a pathological sense—is, after all, tolerant and amenable in a vellous degree.

The question which you might properly put to me is, what did I expect to happen when I left the treacherous ulceration in the caecum clamped at the bottom of the iliac wound? My answer is this, that I hoped that the intestinal perforation would remain securely blocked until adhesions had taken place between the neighbouring coils of bowel and the adjacent parts of the parietal peritoneum; and, I think, this is exactly what did happen. At any rate, here is the boy sound and well, six weeks after having undergone the perils of a general septic peritonitis. There can be no doubt that the operation saved this boy's life. And if you ask me what I consider to have been the chief element in obtaining this gratifying success, I shall say that, in my opinion, it is the thoroughness with which we washed over, and washed out the infected serous membrane with that exceedingly hot saline solution.

NOTES.

A Graphic Study of Tremor.—As the result of an extended study by graphic methods of tremor as observed under varying conditions of health and disease, Eshner ('Journal of Experimental Medicine,' vol. ii, No 3, p. 301) expresses the conclusion that all muscular movements are made up of a series of elementary muscular contractions and relaxations in alternation, which may be appreciated as tremor in conditions of both health and disease. The differences between different tremors were found to be in degree rather than in kind, *i.e.* no one form of tremor appeared to be distinctive of any one disease or group of diseases. No definite relation was found to exist between one form of tremor and any other. The frequency of movement was in inverse ratio to the amplitude, and *vice versa*. Habitual movements were performed with greater freedom from tremor than unusual movements. No material difference could be discerned between the movements of the two sides of the body, except in so far as related to habitual or unusual activity.—*Medical Record*, June 5th, 1897.

THE CLINICAL JOURNAL.

WEDNESDAY, JULY 7, 1897.

TREATMENT OF SIMPLE FRACTURES BY OPERATION.

BY

W. ARBUTHNOT LANE, M.S.

SUFFICIENT time has elapsed since I commenced systematically in 1893 to treat by operation fractures of the long bones, in which I was unable by manipulation, splints, &c., to retain the surfaces of the fragments in accurate apposition, to make me feel that it would be of some interest to you if I reconsidered the subject in the light of the knowledge gained by such experience.

In a paper entitled "A Method of Treating Simple Oblique Fractures of the Tibia and Fibula more efficiently than those in common use," published in the 'Transactions of the Clinical Society' for 1894, I pointed out that—

1. The results of treatment of fractures of the bones of the leg by manipulation and splinting were most unsatisfactory, since the mechanics of the limb were very considerably damaged, and the wage-earning capacity of the labourer often enormously depreciated. The extent of this depreciation varied, of course, with the degree of perfection of control of the lower limb required by the particular trade of the individual. For instance, a steeplejack or fireman would most probably have to give up his employment, while a shoemaker or tailor need experience no ill effects as regards his wage-earning capacity.

2. The results also varied greatly with the age of the individual at the time the injury was sustained; while in young life considerable alteration in the mutual relationship of the axes of the fragments to one another may be met by accommodation changes in the articular surfaces, and by a modified rate of growth of bone in the epiphysal line in accordance with the law I formulated, viz. that *the rate of bone formation in the several parts of an epiphysal line varies inversely with the pressure transmitted through them.*

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After a certain period of life such parts of the opposing surfaces of articular cartilage which, in consequence of this alteration of the mechanism of the adjacent joints, sustain an abnormal amount of pressure, undergo the changes I have fully described under the head of pressure changes as *mechanical or traumatic arthritis*.* These are generally regarded by pathologists as evidence of the presence of a disease called rheumatoid or osteo-arthritis.

3. That in a simple oblique fracture the chief obstacle to replacement of the fragments in accurate apposition is *the hæmorrhage into, and inflammation of the soft parts, which constitute ties in the length of the limb*, with consequent approximation of their points of attachment. In compound fractures in which there is a free exit for the blood there is not the same difficulty in restoring the bone to its normal form.

4. That spasmodic muscular contraction affords a very slight and transitory obstacle to the replacement of fragments. It, such as it is, can be measured and placed in abeyance for the time being by putting the patient under an anæsthetic.

5. That the method of splinting fractures of the lower extremities had, up to that time, been placed upon a false basis from the principles entailed in the application of the vertical foot-piece.

Later I extended my observations to the varieties of fractures which result from excessive forcible abduction of the foot, which are usually described by surgeons as Pott's fractures, and I found that these were followed by results which were, as a rule, more disastrous than almost any class of fracture of the lower extremity. On cutting down on the fragments of the fibula and exposing them freely, and attempting to restore them to their normal relationship by the methods of manipulation described as efficient for the purpose, it was at once obvious that it was impossible to reconstitute the bone in this manner, and that nothing short of the

* "Causation; Pathology of the so-called disease rheumatoid arthritis and of senile changes," 'Trans. Path. Soc.', 1886. "Chronic traumatic arthritis," 'Lancet,' Jan. 30, 1892, and CLINICAL JOURNAL, Feb. 12, 1896.

elevation of the fragments and their forcible apposition by means of forceps associated with the use of a wire suture would bring about the desired effect. Reconstitution of the fibula at once restored the foot to its normal form.

When the internal malleolus was broken off and displaced, the restoration of the fibula to its normal form by operation brought the fragments of the inner malleolus into accurate apposition, and rendered it unnecessary to connect them with wire.

If, however, the lower extremity of the tibia is not restored to its normal form by the reconstitution of the fibula, it can be readily drilled and sutured in a similar manner.

A consideration of the results of fracture of the femur, and indeed of all other long bones in the body, showed me that surgeons were mistaken when they said that they were able to bring the broken fragments into accurate apposition, and consequently to restore the bones to their normal form, by means of manipulation and splinting.

This I pointed out very clearly, and hoped to obtain definite scientific statements supported by some kind of evidence from those who would not accept these views.

My strongest supporter, and a very staunch one too, was Dr. Alfred Parkin, of Hull. I found that his views coincided very closely with mine. He stated them, together with some of the experiences on which they were based, in a letter to the 'Lancet,' April 21, 1894, written in his usual clear and incisive manner. Dr. J. W. Smith also stated what he knew from personal knowledge of the disadvantages which result from the usual treatment of fractures, in a letter in the same copy of the 'Lancet.'

I expected and experienced opposition in two directions. I believed that many surgeons would say that the statements I had made as to the bad results obtained by treatment of fractures by manipulation and by splinting were very much exaggerated, and that they were able to succeed in restoring the broken bone to its original form, and that the mechanics of their patients and their wage-earning capacity were but slightly, if at all, affected in consequence of the accident; also that they would substantiate this by the production of the subsequent history of cases that had been under their care, and which I presumed would be obtained with little difficulty.

In order to meet this objection I made very extensive inquiries of medical men practising largely among the labouring classes, of instrument makers, of large employers of many kinds of labour, of the labourers themselves, and of those who have to do with assisting the poor both inside and outside our infirmaries. To supply my observations by independent evidence, I asked two of my house surgeons, Messrs. Steward and Beddoe, and two dressers, Messrs. Roberts and Clapton, all keen and absolutely reliable men, to see and investigate as many of these cases as they could. These gentlemen kindly did so, and furnished me with most convincing corroborative evidence. Mr. Beddoe put together a number of these cases of Pott's fracture and fracture of the shaft of the femur, and published them in a very carefully written and most interesting paper in the 'Lancet,' June 1st, 1895, under the title "The Treatment of Fractures." Mr. R. P. Rowlands, who as dresser had under his care several cases of fractures, which I had treated by operation, published his views on the subject of Potts' fracture, in the 'Guy's Hospital Gazette,' February 29th, 1896. He has treated the subject in a most masterly, logical and scientific manner; and I would strongly urge you to read it. I can best illustrate the form of objection referred to above by quoting from a lecture on modern treatment of fractures, given by Mr. Marmaduke Sheild, and published in the CLINICAL JOURNAL of May 15th, 1895, and bearing directly on this question, since I have no doubt it represents the views of many surgeons on the subject. Written carefully, chiefly with the object of contradicting the principles involved in my suggestions, it forms a good subject for our study and criticism. He took as the text for his paper the following statement, with which I concluded a lecture on the subject, and which was published in the same journal:—"The treatment of fractures as it exists at present is a disgrace to surgical practice." I will quote freely from this very excellent paper, and ask you to follow me closely, as his statements and arguments seem to me to prove rather than negative my views on the treatment of fractures. He says: "The technical difficulties of dealing with a bad fracture are almost too numerous to mention, but

I will support Mr. Lane when I fearlessly assert that the majority of students and medical men ignore the subject, or give it such scant attention that the ultimate results of fractures are too often deplorably bad. Actions for malpractice in the treatment of fractures are still too common, and a badly united broken leg leading to lameness and deformity is a lasting advertisement of incapacity to the surgeon who is so unfortunate as to be responsible." I do not remember ever having stated "that the majority of students and medical men ignore the subject, or give it such scant attention, that the ultimate results of fractures are too often deplorably bad;" nor do I accept the assumption of cause and effect contained in these words as correct. And again, "If infinite pains and trouble be taken over even oblique fractures, the results are far different from what Mr. Lane would lead us to believe, and the results of private practice treatment by careful surgeons would probably show, in the vast majority of cases, excellent results without any operative interference. I here speak largely from my own experience."

Now as to how this is to be effected is shown by the following extract from Mr. Marmaduke Sheild's lecture, which I quote at length, in order that you may be able to formulate from a careful consideration of it some definite mechanical principles which should guide you in your treatment, which he states has proved so successful in his hands.

"I may now give a sketch of how I believe a fracture should be treated, and will take as an illustration a bad case of oblique fracture of the leg. In the first place I long ago learned from the practice of a distinguished surgical baronet the folly of attempting at once to fix a bad fracture in accurate position. *The 'setting,' as the public persist in calling it, should be postponed until the inflammatory effusion has to some extent subsided, and the blood is beginning to be absorbed. The spasmodic contraction of muscles, so marked when they are at first irritated and partially lacerated, subsides markedly in a week or ten days.* Hence I should place the limb upon a comfortable back splint, with side splints, and sling it in a Salter's cradle. The seat of fracture should be exposed, and a cold lotion, composed of spirit, lead, and water, constantly applied on strips of lint. Bullæ should be at once pricked with a fine needle.

Tight splinting and rigid confinement with bandages should be avoided. In a week or ten days I should, under ether, get the fracture into good position, and this would be greatly aided by having the knee well bent, and making extension from the foot. Splinting should be most carefully carried out, pads being introduced where needful, and the limb should be inspected daily, the most scrupulous care as to application of the splints and position being observed. In a month or five weeks leather splints with straps and buckles should be applied. These must be made by a skilled instrument maker. Towards the sixth week the bones will be firmly uniting, and the limb should be daily taken out of its leather case and gently massaged. The ankle and knee should receive especial attention, being rubbed and moved; as union progresses massage and movement are executed more vigorously. It is especially important that *all movements*, as those of abduction and adduction of the foot, should be carried out. This necessitates the movement in their sheaths of the tendons round the ankle. Hot douches of sea water are also most beneficial. In bad cases massage has to be kept up for at least six weeks to two months, and I affirm very strongly that in the after treatment, which is generally quite ignored, lies largely the successful results or otherwise if bad fractures.

"Should the ankle or knee be stiff, movements under gas should be at once instituted. In a fracture near a joint, such as a bad Pott's fracture, the treatment is essentially the same, except that about the tenth day the foot is encased in a carefully applied plaster apparatus, the patient being deeply anæsthetised and the parts held in proper position by an assistant until the plaster is firm. The foot must be at right angles to the leg, and a little inverted. At the end of the fifth week leather supports are substituted, and prolonged massage insisted upon. In all fractures in the vicinity of the joints, except the hip, I have found the plaster apparatus applied under anæsthetics, after the swelling has subsided, the most advantageous. It will be noted that I have laid no stress upon the oblique foot-piece as so strongly advocated by Mr. Lane.*

"I have had such good results with the vertical

* "Fallacy of the Vertical Foot-piece," 'Brit. Med Journ.,' 1894.

foot-pieces, taking care that the great toe, patella, and anterior superior spine are in the same line, that I see no reason to alter this method. It will be thus seen that I estimate to get a good result, and the most careful and watchful personal care of the surgeon is needed over a period of some two months or more. Besides, I would strongly maintain that much of the success obtainable is due to the mechanical skill of the surgeon, and specially to the celerity and ease of his bandaging, to his care in padding, strapping, and such like small details, of which the least is often the most important."

Though he criticises adversely the various reasons I have given that, in my opinion, render it necessary to operate, and though he allows that owing to the ignorance of the subject by the majority of surgeons, the ultimate results of fractures are too often deplorably bad, he asserts that a good result can be obtained by a skilled surgeon who treats the case carefully for some two or more months. He wisely avoids any statement as to the necessity of bringing the broken ends into accurate apposition, or of restoring the bone to its original form. Allowing the statements to be true, my experience would lead me to conclude that as there are many surgeons who possess in a high degree of development the qualities that would appear to command success in the treatment of these fractures, their frequent failures must be due to their not taking sufficient pains for a sufficient length of time. Now, if the highly accomplished few fail so frequently by the use of such methods, what can the majority hope for? A careful examination of the cases that have come under my care have unfortunately not shown that the result bears any very definite relation to the skill, dexterity, or carefulness of the surgeon.

I must leave you to decide how far in your opinion Mr. Sheild is justified in continuing to apply the principle, or the want of principle, involved in the use of the vertical footpiece, solely on the ground that he fancies he has obtained such good results with it, when it has been clearly shown to be mechanically false, and productive of displacement. It is quite open to him to try to demonstrate that the statements and arguments I used are incorrect, since these observations can be readily made on the living and dead body. Should he succeed in satisfying himself and us in this manner as to the correctness of the mechanics he

advocates, we would willingly accept his evidence as an argument in favour of the use of the particular apparatus; but we cannot accept his statements unless supported by stronger arguments, and some more definite and tangible evidence than he produces.

If we wish to consider ourselves scientific surgeons we must be guided in our practice by well-recognised mechanical principles, and not follow blindly a creed which can be proved by naked-eye evidence to be false, *merely because* we are imbued with the belief that it afforded us such results as in the past we were satisfied to call *good*.*

The second form in which I expected that objection would be taken to operation was on the ground that it is possible to obtain accurate apposition by means of manipulation and splinting alone. Most of those who have criticised my treatment have avoided this line of argument, and have stuck to that illustrated by Mr. Marmaduke Sheild in his paper, namely, that their particular results as regards the patient's well-being and comfort were so good that they could not be improved on by operation. Mr. Christopher Heath, in a clinical lecture "On Fractures of the Lower Limb," published in the *Lancet* of January 4th, 1896, furnishes us with an excellent illustration of this argument, which is put forward with all the knowledge and experience gained by a most able and distinguished surgeon in a large general hospital.

The following remarks are extracted from his lecture:—

"I would remind you how important it is, in fractures of the leg particularly, that the fracture should be set thoroughly and accurately. Of course, I know well that there are many difficulties. *Immediately after the accident all the muscles of the limb are more or less in a state of spasm, and tend, therefore, to pull the bones into abnormal positions; but that state of spasm passes off in the course of a few hours, and you can generally manage with care and patience to put the limb into a proper position, and unless that is done, and done accurately, the surgeon has not treated the case properly.* Looking at this specimen from the

* The word *good* as used in this sense is most misleading. It is really meant to imply *not absolutely bad*. It must not satisfy us now-a-days, and perfection or the nearest approach to it must be our goal. We will accept no compromise.

museum, you can see that there has been a fracture of both the tibia and fibula, and the probability is that they were broken by direct violence, because the fractures are nearly opposite one another, whereas if the accident had occurred from indirect violence the fracture of the fibula would have taken place higher up. I therefore think that we should be right in saying that this was a case of direct violence, causing, as you can see, very considerable displacement."

"The lower fragment of the tibia passing behind the upper fragment, union in this case has been very complete, abundance of callus having been thrown out, but the patient recovered with a shortened and deformed limb. This is just the class of case of which I was thinking when I emphasised the importance of thoroughly setting a fracture. In this case apparently there has been no thorough setting or keeping of the parts in apposition. The first thing to see to in such a case is relaxation of the muscles while extension is applied to the limb. The thigh is flexed, and an assistant should pull on the thigh in the manner shown, the lower part of the thigh being firmly clasped and held perpendicularly to the recumbent body; the leg is then flexed to a right angle with the thigh so as to relax the muscles of the calf, and the surgeon, grasping the foot, can manipulate for fracture or dislocation. It is in this form of fracture of both bones of the leg that I would recommend you occasionally to flex the limb at the seat of fracture if you cannot get the bones into good position. This may seem a dangerous proceeding, but it is not so hazardous as one would think, though you must nevertheless be very careful to avoid making a simple fracture compound. In the average case, however, you can set the fracture in the way I have described without much trouble, and in compound fractures you can manage it with greater ease, and, moreover, you can insert your finger and feel if the two ends are in accurate apposition. In compound fractures also you will be able to thoroughly investigate the presence of a spiculum of bone preventing accurate apposition, and no one would hesitate to push the bone out of the wound and saw off such a projecting spiculum. In cases of compound fracture, where you find, the moment after you have set the limb, that the bones become displaced again, it would be quite within the bounds of good surgery

to divide the tendo Achillis subcutaneously, or to drill the two fragments and put in pegs or screws to hold the bones together; and I may say that this method of pegging or screwing has been recommended by an enterprising surgeon not only for compound, but also for simple fractures. *But I cannot conceive how anybody can believe that it is justifiable to convert a simple into a compound fracture;* and of this I am quite certain, that the majority of surgeons, for the present, will remain content with the usual methods of treatment."

As you see, Mr. Christopher Heath has no hesitation in asserting *now important it is, in a fracture of the leg particularly, that the fracture should be set thoroughly and accurately; and unless that is done, and done accurately, the surgeon has not treated the case properly.* I heartily agree as to the correctness of this teaching, but I would assert without the slightest hesitation that it cannot be brought about by manipulation in the case of simple oblique fractures of the lower extremity. Such fractures form a very large proportion of injuries to the leg. It may be done in compound fractures in which the opening in the skin and adjacent soft parts has been sufficiently large to allow of the free discharge of blood. Curiously enough, both these surgeons, as do surgeons generally, regard *the spasmodic contraction of muscles as being the important obstacle to restitution of the bone to its normal form.* This, however, would exist in at least as great a degree in compound fractures as in the simple ones. The difficulty of replacing the bones in the former is small as compared with that present in the latter. Since Mr. Christopher Heath says that "the state of spasm passes off in the course of a few hours," it can offer but a very temporary obstacle. But I have found that the difficulty experienced in effecting reduction increases rapidly with the inflammatory changes that take place in the soft parts about the fractures. Let us go back to Mr. Marmaduke Sheild's paper, and see what he says about it. These are his words:—"Mr. Lane's second point, that the shortening is due to hæmorrhagic effusion and inflammation rather than to muscular contraction, seems to me hardly proved. *Indeed, I cannot but hold that the contrary is the case,* seeing the remarkable manner in which great shortening and deformity can be reduced under deep ether anæsthesia. This I have witnessed so often that I cannot be

mistaken. Should the parts be left displaced for weeks or months until structural softening of the muscles occurs from the organisation of inflammatory material, I could then understand the immense resistance encountered by Mr. Lane, but this with ordinary careful treatment should not occur. Though muscular contraction may have been overrated in producing the deformity fracture, I feel sure that the consideration and treatment of it in practice is of the greatest importance." If Mr. Sheild does not consider that hæmorrhagic effusion and inflammation are the forces opposing restitution of the fragments, why does he postpone the "setting" until the inflammatory effusion has to some extent subsided, and the blood is beginning to be absorbed? It seems to me that such success as he obtained is due to an unconscious recognition of what I have clearly demonstrated. These two gentlemen appear to have very different views about the manner in which the same cause acts. They both assert that spasmodic muscular contraction is the important obstacle to reconstitution of the fractured bone. How does this knowledge guide them in their treatment? Mr. Christopher Heath considers that "the spasmodic muscular contraction passes off in a few hours," and presumably he postpones placing the fragments in accurate apposition till such time has elapsed, and then does it, as he supposes, effectually and at once.

Mr. Marmaduke Sheild states that "the spasmodic contraction of muscles subsides markedly in a week or ten days," when he proceeds "to get the fracture into good position." It would appear, however, that some factor exerts an influence considerably beyond this period from the constant attention the fracture requires. Now this factor is extravasation of blood and its consequences.

Personally I do not think that the spasmodic contraction of muscles exerts any appreciable influence whatever, and that it may be left out of consideration in our treatment. One has only to expose such a fracture of the tibia and fibula when the patient is deeply under an anæsthetic, and when, therefore, all spasmodic contraction may be assumed to be absent, and to endeavour by traction, elevation, and forceps to bring the broken surfaces into accurate apposition, in order to recognise that there is some other very powerful force which opposes such replacement, and which

requires to be met by a corresponding opposing force. It also bears a direct proportion to the extravasation and inflammation present.

I have nothing to add to what I have already said as to the causation and treatment of fractures, and especially of those of the lower extremity.* My more extended experience has served to prove still more thoroughly the correctness of the attitude I took up as to the wisdom and safety of operative interference.

Perhaps a few hints of the details of treatment of several forms of fracture may be of service to you. Elevators, sequestrum, and lion forceps, combined with tractors for manipulation, will be



Fig. 1.



Fig. 2.

found the most suitable and effective means for bringing the fragments into accurate apposition, and for retaining them there till you have connected the bones. A modification of the ordinary lion forceps has been devised by Mr. Peters (Figs. 1 and 2). It is of such a form that holes may be drilled through the blades in almost any direction, obviating the difficulty one so often experiences of finding a suitable place other than that beneath the blades of the forceps, where the

* "Some Remarks on the Treatment of Fractures," 'Brit. Med. Journal,' April 20th, 1895. "Clinical Lecture on Fractures," 'Guy's Hospital Gazette,' July 20th, 1895.

bone can be perforated by the drill, and through which the screw or wire can be passed to the best advantage. In fractures of both bones it is hardly ever necessary to expose the fibular fragment, as the ends of that bone usually come into position when the tibia has been restored to its normal form. If they do not come into perfect apposition it is not a matter of very great importance if the fracture is situated in the upper half of the bone, since the mechanics of the ankle-joint are not appreciably

extremity, which must be made to enter and fit accurately a corresponding angular interval in the opposing fragment. We are asked to believe that this can be effected by various manipulations on one occasion by some surgeons, or by a combination of methods of an ill-defined character extending over a long period of time by others. Those who operate in these cases will find that to secure such accurate coaptation of surfaces as will restore these bones to their normal form will very often make

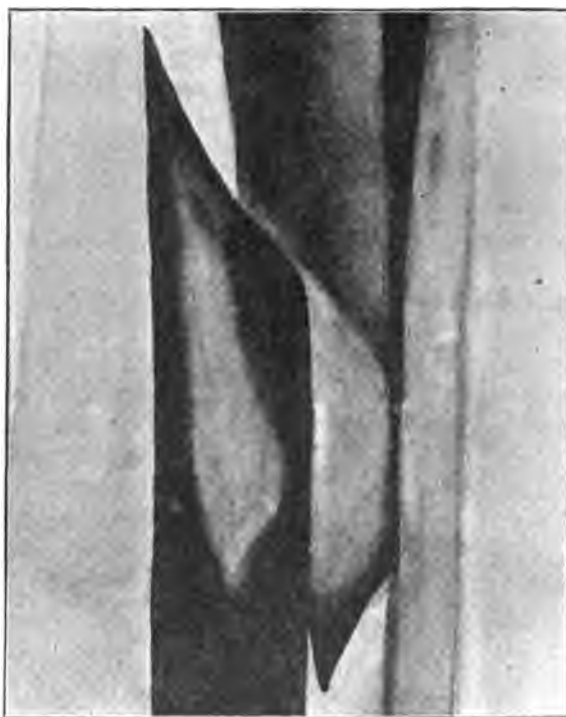


Fig. 3 is a skiagram made for me by Messrs. Watson and Sons, of High Holborn, and shows very well the spike-like extremities of the fragments, and the angular intervals in which they must be returned. The bones were placed in as good position as was possible, and were fastened securely on a splint. A careful study of this fracture will serve to convince one not only of the impossibility of restoring the bone to its normal form by manipulation and splints, but also of the *great difficulty* which is frequently experienced in doing so when the fragments are freely exposed by an operation.

altered by a slight variation in the axis of the fragments so high up in the shaft. If, however, the fibula be fractured in its lower fourth, and its continuity is not established when the tibia is reconstituted, it would be necessary to cut down upon it also.

The direction of the fracture through the tibia varies with the mode in which it is produced. When it results from indirect violence it is very oblique, each fragment often presenting a V-shaped

quite as great a demand on the skill of the surgeon as any operation in surgery, and I believe that the difficulty which is frequently experienced will prevent operative interference in this particular form of fracture from coming into very general use. When produced by direct violence, fractures do not present the same characters, being much less spiral or oblique. They are usually much easier to reduce when the ends of the fragments are exposed, but without operative measures

it is almost always impossible to reconstitute providing the fracture be *simple*, if the fragments overlap, owing to the greater amount of hæmorrhage and laceration of the soft parts which are associated with this form of violence. If the fracture be *compound*, and blood has escaped freely from the wound, the same difficulty need not be experienced. The incision I find most generally useful is one along the anterior margin of the tibia supplemented by a transverse one running backwards from its centre, and crossing the fracture about the middle. By means of this both the inner and outer aspects of the tibia can be seen sufficiently to enable the surgeon to get at them with his instruments. You will find that the lower fragment of the tibia is almost always displaced upwards, outwards, and backwards behind the upper. Having got the surfaces into accurate apposition, the surgeon must consider whether he can retain them best by screws or by wire. The screws I use are those made for me by Messrs. Down Bros., or failing them the ordinary carpenters' screws, and the wire is of virgin silver heated to a red heat, so that it is softened and can be bent more abruptly than the original wire. In this manner



Fig. 4.

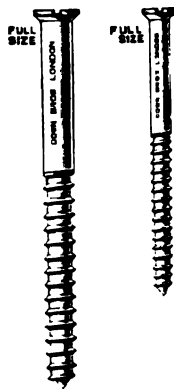


Fig. 5.

separation of surfaces due to the presence of a curve in the wire is avoided as much as possible. In order to diminish the number of drills and rimers necessary if carpenters' screws be used, to reduce the difficulty of introducing the screw, and the likelihood of fissuring the compact tissue, and to facilitate the operation generally, Messrs. Down Bros.

made for me screws of various lengths from 1 to 2½ inches, and of two gauges. They differ in construction from the ordinary wood screw, although of the same type, being made of steel with a smaller barrel and head, the edges of the latter being rounded off. They are silver-plated to prevent their rusting. The awls for perforating the bone are made in two sizes, one for each gauge of screw. There are also two sizes of rimers to enlarge the opening to carry the shank or barrel of the screw. It is hardly necessary for me to call attention to the importance of riming the aperture in the proximal plane of compact tissue carefully to avoid splitting of the bone and consequent loss of approximating power resulting from a lack of support at the point from which the head takes its bearing. As a rule it is safer to use wire than a screw in these fractures of the tibia, for the reason that unless the greatest care is taken in using a drill of suitable size, and in riming the proximal layer of compact tissue to fit the barrel of the particular screw, fissuring of the fragments may result. It would seem advisable, before the surgeon uses a screw for this purpose on the living subject, that he should make himself thoroughly familiar with its mechanics and with its mode of application in the recent bone. In some cases the fragments are found to be fissured or comminuted, and in such application of the screw is often very difficult. The surgeon must remember that the screw is by far the best means by which the surfaces can be retained accurately and immoveably in apposition; and that if there is a very great tendency to separation of the fragments, he must make use of it if possible, either alone or in combination with wire. If he cannot use a screw, or if, once apposition has been secured, there is then little tendency to displacement owing to accuracy of fit, he should employ wire. The surgeon must not expect that any of what goes by the name of *callus* will form about the fracture of a bone treated successfully in this manner. It is not deposited about the broken ends when the bone is restored to its normal form, and the fractured surfaces are retained in firm and accurate apposition. The presence of callus indicates imperfect apposition or a want of complete fixation, both evidences of an unsatisfactory surgery. It is very useful in the savage, and in the lower animal, but is a dis-

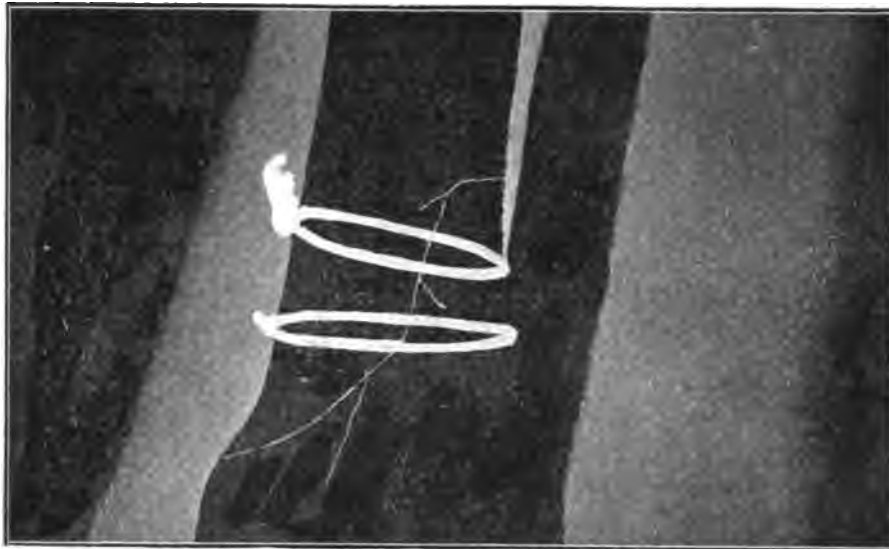


Fig. 6 represents a recent oblique fracture through the tibia, produced by direct violence. There was considerable displacement of the fragments upon one another; but when the broken surfaces were brought into accurate apposition, their irregularities opposed upward displacement, and rendered it possible to connect them securely with a couple of silver wires. In such a fracture as is represented in Fig. 3, owing to the strain exerted by the ties in the length of the limb, and the absence of any irregularity of the outline of the margins of the fragments, the use of the screw for forcible and accurate apposition of fragments and their subsequent retention is absolutely necessary.



Fig. 7 represents a very oblique or practically spiral fracture of the tibia, and presumably an equally oblique fracture of the fibula at a higher level, resulting from indirect violence. The patient was a coal-heaver, and the injury was sustained more than two years ago. The tibial fragments were got into accurate apposition, and were retained there by means of the screw which is represented in position. Although it has been there for so long it shows no obvious change. He has suffered no pain or discomfort whatever, and he carries his load of two hundredweight as well as ever.

advantage in members of a civilised community, since it can only exist with unskilled work.

In the group of injuries comprised under the term Pott's fractures the surgeon will find that unless the fracture runs through the attachment of the interosseous ligament the lower fragment cannot be materially influenced by such movements of adduction of the foot as are made use of for the purpose by surgeons generally, and the broken

established. Screws are very rarely required for this form of fracture, since, the tibia being intact, the tension of the ties in the length of the bone is inconsiderable, so that there exists comparatively little force tending to produce overlapping of the fragments. The condition of the ends of the bone must determine the surgeon as to whether he perforates the fragments or encircles them with wire, or adopts both these measures. In fracture of the



Fig. 8 was a similar case that was operated on about the same time as the preceding, and gave me great trouble in effecting perfect apposition.

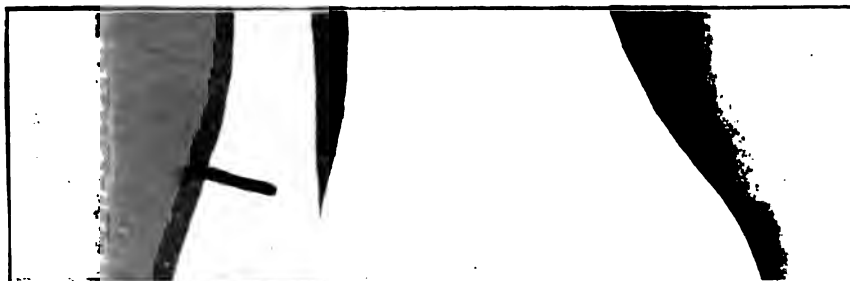


Fig. 9 shows a case of Pott's fracture, in which the fibula was restored to its normal form, the fragments being secured firmly by silver wire.

surfaces certainly cannot be brought into apposition by them. The fragments should be freely exposed, and by means of elevators and sequestrum forceps the surfaces are brought firmly and accurately together. The difficulty in effecting this varies considerably with the length of the fracture, since the longer the fracture the more difficult the process of coaptation, though the reverse applies to the difficulty of retaining them once apposition is

femur wire is usually sufficient, but the surgeon must be guided by the same mechanical principles as in the case of the fractures of the tibia and fibula.

There is one form of fracture for which a screw is alone suitable, and that is a fracture of the olecranon. Here a screw driven into the base of this process secures perfect apposition, and restores to the joint its normal functions. Care must be

taken to embed the head of the screw, otherwise it may have to be removed months afterwards. This can, however, be effected through an incision whose length equals the diameter of the head of the screw. I have not found it possible to secure accurate apposition of these fragments by wire. In fractures of the clavicle, acromion, radius, and ulna, wire affords an efficient means. I have not operated on a simple fracture of the humerus, but in the case of ununited fractures I have succeeded in obtaining bony union by means of the screw, when others had failed with wire on more than one occasion. This is due to the difficulty of preventing movement of the ends of this bone on one another by means of wire when the fracture is nearly transverse. It is probable that the screw would do best for this bone. For fractures of the lower jaw, which are almost always compound, I have usually used wire, but have on several occasions found a screw of service when wire had failed. This was particularly the case when the operation was done for non-union some weeks after the fracture had been sustained. This is due to the body of the bone softening at the seat of fracture, then readily breaking or crumbling when perforated with the drill. In simple fractures I always try and keep any loose fragments, fixing them securely and carefully into position. I feel that I cannot condemn too strongly the mutilation of the fragments suggested by Mr. Christopher Heath when he says, "In compound fractures also you will be able to thoroughly investigate the presence of a spiculum of bone preventing accurate apposition, and no one would hesitate to push the bone out of the wound and saw off such a projecting spiculum." The fracture should be freely exposed, and the spiculum placed accurately in its corresponding recess in the other fragment.

In one case of fracture dislocation of the spine I employed wire to retain the bodies of the vertebræ in their normal position, having reduced the dislocation.* The wire was passed round the adjacent spinous process, and this succeeded for a time, but the wound having become foul owing to some carelessness, the wire got loose and ceased to serve its purpose. In another case of this sort I should perforate the bases of the spinous pro-

cesses to prevent the possibility of the wire slipping.

Risk of infection of the wound in the hands of a competent surgeon is small, while the benefit which the patient derives from the operation is enormous. Even if the wound does become infected, it may be necessary to remove the screw or wire, perhaps even with a small sequestrum, after they have served the purpose of retaining the fragments in apposition for a length of time sufficient to allow of their union. This happened to me in a very difficult case of fracture of the tibia and fibula, the patient, however, recovering with a perfectly useful limb.

It seems to me quite time that surgeons should throw off their prejudices, and let their actions be guided by facts and reason instead of by tradition and superstition. If they are still under the impression that the statements and teaching of the present day are correct, they have ready at hand the cases they themselves have treated. These can be skiagraphed, and reliable evidence obtained both as to their condition and as to the mechanical well-being of the patient. Let them supplement their arguments and statements by the production of their own cases, and I shall always be pleased to place those I have operated on at the disposal of anyone who is sufficiently interested. In this way we shall be more likely to arrive at the truth than by any amount of discussion. It is a curious thing that while a surgeon possessing any confidence in his methods will not hesitate to lay open the joint in the case of fracture of the patella, remove the blood effused, and bring the surfaces of the bone into accurate apposition by means of wire, yet the same surgeon would entail great pain, distress, and financial depreciation on the unfortunate patient affected by the so-called Pott's fracture, because *he says it is unjustifiable to convert a simple fracture into what he incorrectly calls a compound one.* Why is this? The answer is an easy one. It is merely an example of the unreasoning imitative capacity of the human animal. Certainly, in my own experience, the depreciation resulting from Pott's fracture is usually much greater than that following a fracture of the patella treated by splints. Personally I should have imagined that of all simple fractures the operative treatment of that of the patella is accompanied with more risk to life and limb than that of any other bone in the body.

* "Fracture (Dislocation) of Spine; Reduction; Temporary Recovery," 'Lancet,' September 17th, 1892.

Judging from the popularity of all kinds of unsatisfactory and unscientific compromises, undertaken with the intention of not exposing the joint, I am convinced that accidents are constantly happening owing to some want of precaution somewhere. These imperfect methods can only be suggested by "caution," which is often synonymous with "fear," gained by experience in the school of adversity.

The surgeon is, however, only too ready to attempt by means of operation to improve the mechanical condition of patients who have suffered from his want of energy, knowledge, and skill.

Take, for instance, the limited movements or the ankylosis of the elbow-joint which results so often from fracture about the lower epiphysis of the humerus in the young subject. To give such a patient a movable joint the surgeon does not hesitate to cut away large portions of the humerus, ulna, and radius, to expose the patient to considerable risk, and to the endurance of great pain for many weeks, in the hope of his obtaining, after a considerable period of time, an arm much shorter than its fellow, and as a mechanical arrangement almost invariably vastly inferior to it. Can anything be more brutal, illogical, and unscientific than all this? Fortunately, skiagraphy has placed in our hands, and in those of our patients, a means of determining the condition of parts at the time the injury is sustained, so that the surgeon has no longer any excuse for deluding himself into the belief that he has replaced the damaged structures in their normal position. In this particular fracture the bone *must* be restored to its original form if the elbow-joint is to perform its functions in a normal manner. To attack a difficult fracture of one or more bones may make a certain demand on the confidence of the operator in his capacity to ensure asepsis, not only in himself, a comparatively easy task, but also in those who assist him. It is, however, no more than is called for in other important operations in which the skin is intact previous to the incision.

The difficulty experienced by those operating for the first time is that the method is a comparatively new one to the profession, and an accidental infection of the wound, which in other every-day operations would call for no comment, might do so in this case. A little familiarity with the procedure will, however, serve to remove this. The

public will in future exert a powerful influence on the treatment of fractures. Curiously enough they, in common with the majority of the profession, share the belief that it is the duty of the surgeon to restore the broken bone to its original form, and in one way and another they have acquired a general knowledge of the form of bones. Possibly the law courts, aided by the skiagraph, may stimulate our action somewhat in this matter.

A CLINICAL LECTURE

ON A CASE OF

ADDISON'S IDIOPATHIC ANÆMIA.

BY

HENRY WALDO, M.D., M.R.C.P.,

Physician to the Bristol Royal Infirmary.

I PROPOSE to lay before you the current views relating to anæmia. It is usually accompanied with pallor, chiefly of the face and visible mucous membranes; but this is not always the case, and unless you examine the blood you may be misled. Remember, too, that pallor is associated with fright, and also with nausea; and that some people are naturally pale from an inherited peculiarity without being anæmic. I do not wish to say much of secondary anæmias, as from hæmorrhage, long-continued drain on the albuminous materials of the blood (as in suppuration, Bright's disease, prolonged lactation, rapidly growing tumours, or from inanition), or of toxic anæmia, as in cancer, syphilis, malaria, among organic poisons, or from lead, mercury, and arsenic among inorganic substances, or the anæmia of pyrexia; but to principally direct my remarks to primary anæmia, as we find it in chlorosis, progressive pernicious anæmia, and in splenic anæmia. A profound anæmia may be associated with pregnancy and parturition, and with atrophy of the stomach, as well as with the presence of parasites (the *Anchylostomum duodenale* and the *Bothriocephalus latus*), in which it may be difficult to distinguish clinically from Addison's idiopathic form (progressive pernicious). You are very familiar with cases of chlorosis, occurring as it does in females at puberty. The patient's skin

is of a yellow-green tinge, from which its name is derived, and it is known to the laity as the green sickness. At the present time I have a patient in the infirmary with progressive pernicious anæmia, and I will now dilate upon some of the symptoms of this illness. Authors are divided as to whether this disease occurs oftenest in men or women. The cases I have seen have been mostly in women. Its attack usually begins at middle age and early advanced years, but it has been known to occur at any age. The patient at present under my care is aged 25. She has been married three years, and she states that her present illness began soon after marriage. Her father is a Portuguese, and her mother English, and her skin is naturally of a dark colour. In all the cases I have seen the skin has been of a lemon-coloured tint, but in this woman it is masked by the pigmentation. In other cases I have observed this lemon tint to vary from day to day in intensity, and it is quite distinct from the yellow tinge of jaundice, in which condition the conjunctivæ are also affected. The lemon tinge is supposed to depend upon the destruction of blood-corpuscles. This patient's lips, gums, and tongue are very bloodless-looking. She has not menstruated for two years. She says she is much thinner than she used to be, and you will find her muscles are very flabby. The muscles are generally found more flabby than wasted. Increasing languor is a symptom which attends this condition, and which sometimes becomes extreme. The pulse, as in this case, may be somewhat large, but it is rather soft and compressible; and if you hold up the arm you will find that it has the water-hammer character of aortic leakage, although there is no other evidence of the blood falling away into the left ventricle through the aortic valves. I tried to obtain a capillary pulse, and failed to produce any redness by moderate friction, but with a glass slide upon the lower lip it was quite distinct. A systolic murmur, soft and blowing in quality, is audible all over the front of the chest; Balfour's theory is that it is probably produced at the mitral orifice by relative insufficiency of the valves in the dilated condition of the ventricle. It is called a hæmic murmur, and is usually loudest over the pulmonary valve area. In this case the murmur is systolic and loudest and rougher over the aortic area. No doubt the vibrations which reach our ears are those

of the walls of the vessel or of the chamber of the heart which is thrown into vibration, and not of the contained blood. At the same time, as Pye-Smith says, we still seem to need some further physical explanation of the undoubted fact that in cases of anæmia murmurs are readily produced and are unusually loud. Palpitation of the heart is very easily set up, and with the visible throbbing of the large arteries annoys the patient. Directly the head is on the pillow loud noises, too, are frequently complained of, and interfere with sleep;—they are noises of approaching locomotives or the ringing of bells, or various other sounds. Slight oedema of the ankles are sometimes found; this patient says she gets it if her legs hang down. An uncomfortable feeling of faintness or breathlessness or giddiness is often present, especially upon the slightest movement. This patient came in vomiting, which she says has troubled her a good deal for the last year. Nausea and vomiting are usually associated with pernicious anæmia. Diarrhœa may also be a prominent symptom all through the disease. This patient was only free from it in the earlier part of her illness. Pyrexia is a variable symptom; it is usually irregular, and you get a normal temperature for a week or so, and then pyrexia may develop and last for another week. This may be the order of events during the time the illness lasts. In examining the blood I advise you to puncture with a surgical needle on the dorsum of the hand just above one of the nails. The red blood-corpuscles are always reduced in number. In my case there are only 760,000 per cubic millimetre instead of 5,000,000. This is an exceedingly low percentage, although there is a case recorded by Quincke in which they were reduced to 143,000. The hæmoglobin represents 20 per cent., and so is relatively increased. In all other forms of anæmia the percentage of the hæmoglobin exceeds that of the red blood-corpuscles, and this becomes a cardinal point in the diagnosis. Microscopically the red cells present a great variation in size, and there can be seen large giant forms, megalocytes, which are often ovoid in form—they are always present. There are also small round cells, microcytes, and of a deep red colour. The corpuscles show a remarkable irregularity in form, elongated and rod-like or pyriform. To this condition of irregularity Quincke gave the name poikilocytes. Nucleated red blood-corpuscles are

also almost always present. They are of the normal size of a red cell, and are called normoblasts. Very large forms of nucleated red cells are called gigantoblasts; these resemble somewhat the larger megalocytes, but they possess a palely staining nucleus. Red corpuscles with fragmenting nuclei are also common in pernicious anæmia.

The leucocytes are usually normal or perhaps diminished in number; and in the graver cases a marked increase in the small mononuclear forms, with a diminution in the polynuclear leucocytes, is often noted. The changed form and size of the red blood-corpuscles may be present in all forms of anæmia with the exception of the large forms of nucleated red cells, the gigantoblasts of Ehrlich, which are only observed in pernicious anæmia.

The best method of fixing and staining blood films is that suggested, from the laboratory of the Royal College of Physicians, Edinburgh, by Dr. Lovell Gulland. The process is as follows. A small drop of blood, drawn in the usual way, is taken up on the centre of a cover-glass held with forceps, and distributed evenly between that and another cover. The utmost care must be taken to avoid all pressure. The covers are then gently and rapidly slid off one another, and dropped with the wet side downwards into the fixing solution. This is made up of—

Absolute alcohol saturated with eosin 25 c.c.
Pure ether 25 c.c.
Sublimate in absolute alcohol (2 grms.
to 10 c.c.), 5 drops (more or less).

A good plan is to keep the three liquids in different bottles, and make up the required amount in the above proportion just before using it. The fixation of the elements is practically instantaneous, but the cover-glasses should be allowed to remain in the solution for at least three or four minutes, to fix the film to the cover. They are then taken from the solution by forceps, and washed rapidly but thoroughly by waving them to and fro in a small basin of water. They are then stained for one minute (not longer) in a saturated watery solution of methylene blue, and again rapidly washed in water. Next they are quickly dehydrated in absolute alcohol, which at the same time removes the excess of methylene blue, cleared in xylol, and mounted in xylol balsam on a slide. The red corpuscles are stained pink, nuclei a deep blue, the bodies of the leucocytes in varying shades

of pink; the eosinophile and basophile granules in the leucocytes are well brought out; the blood plates are stained a fainter blue than the nuclei, and organisms are also well stained.

It has been stated that the alkalinity of the blood is diminished in pernicious anæmia and in chlorosis. The specific gravity of the serum in pernicious anæmia has been found increased and diminished. The proteids are diminished.

Hæmorrhages are common in the retina as well as in mucous and serous membranes, and on the skin. Stockman suggests that repeated small capillary hæmorrhages (these he attributes to fatty degeneration of the vessels)—chiefly internal—play an important rôle in the causation of the disease, which also explains, he holds, the existence of a great excess of iron in the liver. The urine is usually of a low specific gravity and a deep sherry colour, which has been shown by Hunter and Mott to be due to great excess of urobilin.

As regards ætiology, Hunter says that in pernicious anæmia the blood is destroyed in the portal circulation, and that the excess of iron deposited in the liver is the result of this destruction. The agents of the destruction he believes to be ptomaines produced by micro-organisms in the intestines. He receives support from Dr. Hale White, who has shown that a large proportion of cases of pernicious anæmia have had marked gastro-intestinal symptoms. But to prove this it would be necessary that these intestinal symptoms should at any rate occur at an early part of the illness. I cannot say that this was so in any of my cases, and the patient at present in the infirmary states that her diarrhoea has only existed for five months. But pernicious anæmia may not always be one and the same disease. It is quite possible that its symptoms may be due to more than one cause. The ætiology of chlorosis is also very obscure. Virchow believed that a congenital narrowness of the aorta had some causative connection with it. Excessive acidity of the gastric secretions is said to be a cause. Proptosis of the stomach and gastric ulcer often attend cases of chlorosis, and as a possible cause of these conditions tight-lacing would have to be considered. Habitual constipation with retention of fæces, and septic processes leading to absorption of ptomaines or other toxic substances, have been thought by the late Sir Andrew Clark to account for chlorosis.

But the morbid process in chlorosis appears to be a defect in the formation of the corpuscles (deficient hæmogenesis), while in pernicious anæmia there is alone a rapid destruction of corpuscles (increased hæmolysis).

In considering the morbid anatomy of pernicious anæmia you will find the body rarely emaciated. A lemon tint of the skin is present in the majority of cases. The heart is usually large, flabby, and empty. The muscle substance of the heart is intensely fatty, and of a pale light yellow colour. In no affection do we see more extreme fatty degeneration. The mucous surface of the stomach may be atrophied. There is nothing abnormal found in the lungs. The liver may be enlarged and fatty. The iron is in excess, and in striking contrast to cases of secondary anæmia. The spleen shows no important changes, but the iron pigment is usually in excess. The amount of iron pigment is increased too in the kidneys, chiefly in the convoluted tubes. The lymph glands may be of a deep red colour. The bone marrow is usually red in colour, resembling that of a child, and showing great numbers of nucleated red corpuscles, especially the larger forms called by Ehrlich giantoblasts. Since the bone marrow is a former of red blood-cells the change is thought to be rather an effort at restoration than an agency for ill. Changes in the ganglion-cells of the sympathetic have been reported on several occasions. Lichtheim has found sclerosis in the posterior columns of the spinal cord in some cases of pernicious anæmia. In our patient the knee-jerks are normal. The pupils act to light, and there are no pains, so that clinically there is no evidence of this.

In forming a prognosis in these cases the presence of numerous normoblasts appears in some instances to be indicative of an active regeneration in the marrow. Cases in which a majority of the nucleated red corpuscles are giantoblasts are generally more malignant. A marked relative increase in the small mononuclear leucocytes appears to be also an unfavourable sign.

Splenic anæmia, or what French writers call primary splenomegaly, is of the chlorotic type. There is no leucocytosis. The spleen is of large size, and presents a kind of sclerosis or fibrotic condition, consisting in atrophy or sclerosis of the Malpighian corpuscles, and considerable fibrous

thickening of the trabeculæ throughout the organ. The liver is usually normal or slightly enlarged. The blood-cells are sometimes deformed (poikilocytosis), and there may be microcytes. The disease is not a form of pernicious anæmia. Banti is disposed to regard splenic anæmia as a pure splenic form of pseudo-leukæmia, or Hodgkin's disease.

Pernicious anæmia may be simulated by profound secondary anæmia of cancer of the stomach or other parts, and may be puzzling; but the skin is rarely, if ever, lemon tinted, and the blood has the characteristics of chlorosis. You must not mistake the anæmia of the early stage of pulmonary tuberculosis for chlorosis or pernicious anæmia. Syphilis, too, may cause a large amount of anæmia, perhaps by affecting the bone marrow.

The treatment this patient is receiving is rest in bed. All the fresh air and sunlight procurable is beneficial in these cases. She is unable at present to take anything but fluid foods, and chiefly milk (peptonised), and a little wine. If I am in doubt as to the form of anæmia I prescribe iron, and wait a week or so for the result. But cases of pernicious anæmia do not respond to iron. The most beneficial drug seems to be arsenic in increasing doses. It may cause puffiness of the face or pigmentation of the skin, or even multiple neuritis, so that it must be watched. She also inhales oxygen gas three times a day without the least difficulty. Extracts of bone marrow have been recommended, but they are not always well taken. I am giving the red marrow in tabloid (gr. iss).

Acting upon Hunter's theory of the ætiology of pernicious anæmia, intestinal antiseptics have been given. Benzo-naphthol in 10-grain doses every four hours has the advantage of being tasteless. I have also tried animal charcoal, which tends to check the diarrhœa. The beneficial effect of arsenic is said to be owing to its antiseptic properties, and its power over some skin diseases has been explained in the same way. Transfusion of blood has been resorted to in some cases. If there is a suspicion of syphilis you may do good by giving antisiphilitic remedies. You must examine the blood periodically, and remember that cases of pernicious anæmia are very liable to relapse.

NOTES.

Extirpation of the Rectum.—Dr. Byford gives the following advantages of the vaginal method :—1. The vagina can be made to take the place of the extirpated portion of the rectum. 2. The excision can be done as high up as by the sacral method, and with less traumatism, and, in case the peritoneal cavity is opened, with less danger. 3. An intra-peritoneal exploration of the tissues about the rectum can be made before disturbing the rectum. 4. If the operation has to be abandoned after the incisions are made, the wound is less formidable and in a better place. 5. The patient is more comfortable after the operation than after the sacral methods.—*Annals of Surgery*.

Acute Oedema of the Lung.—A recent session of the Paris Académie de Méd. was devoted to this subject, Huchard ascribing it to aortitis, or peri-aortitis, shown by the diminished blood-pressure. The treatment, therefore, should be based on the pulmonary hypertension, the disturbances in the pulmonary innervation, and the renal impermeability, as it frequently accompanies interstitial nephritis. The heart must be strengthened and its work lessened, which can best be accomplished by a large general or local venesection. With this the use of caffeine and camphorated oil should be advised, and strychnine administered internally or in subcutaneous injections. Morphine is strictly contra-indicated owing to its depressing effect. Iodine medication should be immediately suppressed. Death ensues in a few minutes in the violent form, but there may be several attacks before the fatal ones in the subacute form. It should be distinguished from syncope, stenocardia, alimentary toxic dyspnoea, asystolia, uræmia, and asthenia.—*Bulletin*, April 27th.

REVIEW.

A Manual of Infectious Diseases. By E. W. GOODALL, M.D.Lond., and J. W. WASHBOURN, M.D.Lond. London: H. K. Lewis. (Pp. 368. Price 15s.)

Drs. Washbourn and Goodall are to be congratulated, not only for the excellence of their work, but also for the impartial methods they have

pursued in advocating their particular views—views which must be received with respect, coming as they do from two such well-known workers in clinical medicine and bacteriological research. The book supplies a long-felt want, and the new spirit of scientific diagnosis and treatment has prepared for it a very ample and widespread sphere of influence. The volume is intended chiefly for students for whom attendance in the wards of a fever hospital is now compulsory, explaining the positiveness of the statements, which, however, in no wise detracts from the utility of this volume to men in practice. The authors give precise and sufficient directions for the cultivation and staining of bacteria. The teaching in bacteriology is necessarily brief (on account of the limited size of the book), but it nevertheless stands out as a very distinctive feature. With regard to the ordinary specific fevers much valuable information is given for the purpose of arriving at an early and accurate diagnosis, the importance of this being duly insisted on. From a clinical point of view specific fevers are excellently treated. On reading Chapters I to IV, dealing with fever, contagion and infection, and disinfection, one is struck by the care and skill with which the great discoveries on these subjects have been brought into a small compass. The ever-recurring difficulty in private practice of making an early and precise diagnosis of sore throats and rashes is ably discussed. A concise description of the most striking points in regard to each of the rashes is given, together with a diagrammatic representation of the distribution of the rash. These diagrams will be found very valuable and helpful. At the present day diphtheria has gained especial interest from the anti-toxin treatment, and the authors give a scientific description of the nature of the complaint; the commencement of the serum treatment is advised as soon as possible, irrespective of the severity of the case. In considering the question of vaccination, the views expressed are lucid and explicit. Forty pages are devoted to enteric fever. Doubtless only the want of space has forced the authors to comparatively briefly deal with so important an affection. There are some valuable appendices which are well worth attention. In a future edition, cholera and glanders will no doubt be included. The book is well printed and excellently illustrated.

THE CLINICAL JOURNAL.

WEDNESDAY, JULY 14, 1897.

A CONTRIBUTION TO THE SURGERY OF HYDRONEPHROSIS.

BY

J. BLAND SUTTON, F.R.C.S.

ON several occasions I have drawn attention to the fact that many large examples of hydronephrosis, in which the surgeon finds no adequate cause for the distension, there are good reasons for the belief that it began during intra-uterine life. The four following cases bearing on this matter have come under my notice during the last twelve months.

CASE 1. *Large intermitting hydronephrosis due to an inadequate ureter.*—In 1895 Mr. Lennox Browne kindly placed under my care a lady with a large tumour occupying the right half of the pelvis. For many years she had complained of pain in the side, but had only been aware of the tumour for about a year. Physical examination revealed the existence of a myoma in the uterus, and it seemed extremely probable that this tumour presenting above the brim was a pedunculated uterine myoma. As the patient intended to spend some months in Algeria, operative interference was postponed. Three months later, to the patient's astonishment, the tumour disappeared completely in one night, and on her return to England I was able to satisfy myself of the fact, but on examination I found a uterine myoma as big as a cricket-ball lying in the pelvis. I came to the conclusion that the large tumour which was present in the pelvis at my first examination was an ovarian or parovarian cyst which had ruptured.

In February the patient, whilst in Switzerland, was seized with pain in the loin. She sought advice, and on examination, a doctor at Montreux detected an enlargement of the right kidney and recommended her to return to me.

The large cyst which I found at the first examination had now returned, and the history left

no doubt that it was an intermitting hydronephrosis, perhaps due to pressure from the myoma.

In May, 1896, I performed cœliotomy, and found a myoma in the fundus of the uterus, producing retroflexion of the organ; as it was causing no trouble I merely straightened the uterus. A large retro-peritoneal cyst filled the right iliac fossa. This I made out to be a hydronephrotic kidney; its lower circumference reached as low as the fundus of the uterus. The left kidney was

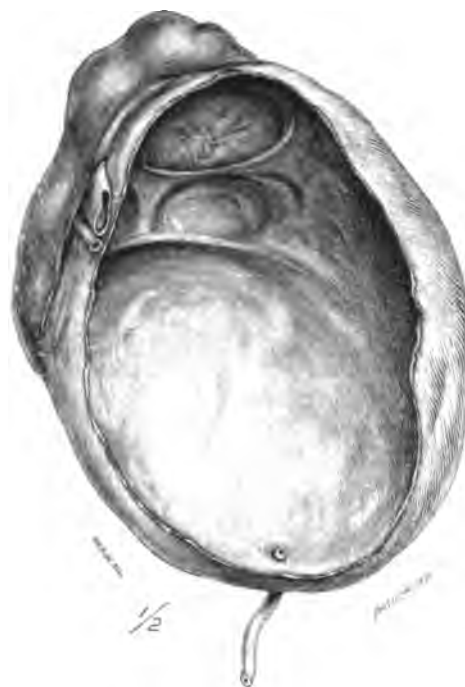


Fig. 1.—Large intermitting hydronephrosis due to an "inadequate" ureter.

normal in size and position. After closing the abdominal incision in "triple layers" by suture, I exposed the right kidney by means of an incision in the right ilio-costal space, and then carefully enucleated it. The elongated slender arteries were secured with silk ligatures, and the wound drained and closed. Convalescence was quick and good. The cortical substance of the kidney was represented by a mere shell of tissue, but the renal pelvis was dilated into a huge sac;

on the floor of this sac there was a minute dimple leading into a narrow (inadequate) ureter, as represented in Fig. 1.

Recovery was rapid and complete; a year later the patient reported herself to be in excellent health.

CASE 2. *Moveable hydronephrotic kidney; inadequate ureter; nephrectomy.*—Mr. A. W. Williams placed under my care a young woman on account of a moveable swelling in the right side of the belly. Its shape, and the readiness with which it slipped under the costal arch, made it clear that the swelling was a kidney. The patient told us also that the swelling varied greatly in size; sometimes it would be large and painful, then she would lie down, and in the course of a few hours the swelling generally subsided, and she would void a larger quantity of urine than usual. Subsequent to such an attack the swelling would not be obvious for many days, and she would be free from pain.

Such a characteristic history made it clear that we had to deal with an intermitting hydronephrosis.

Through an incision in the right linea semilunaris I ascertained the condition of the left kidney and the extent of hydronephrosis in the right one. The right kidney was exposed through an incision in the loin, and the degree of sacculation was so great that I deemed it prudent to remove the organ. On examination of the relation of the pelvis to the kidney, the ureter was found to be as "inadequate" as that represented in Fig. 1.

The patient made a quick and satisfactory convalescence.

The sequence of events preceding nephrectomy in this case are instructive. For some years she complained of "pains in the side," which were regarded as of gastric origin; in due course the moveable swelling appeared in the belly. It seems to me that the attacks of pain were caused by the distension of the renal pelvis, and the increased weight due to the accumulation of urine dragged the organ downward until it became obvious as a "swelling" in the right iliac fossa.

The undue narrowness or "inadequacy" of the ureter was probably a congenital defect.

CASE 3. *Ante-natal hydronephrosis of a single (unsymmetrical) kidney.*—The specimen represented in Fig. 2 was obtained from an infant which survived

its birth a few days. The right kidney only was present; it occupied its normal position in the loin. Its infundibula, pelvis, and ureter were widely dilated, and at the point where the ureter opened into the bladder there was a small circular diaphragm-like valve, but this structure offered no obstruction to the flow of fluid from the ureter into the bladder when tested after death.

The bladder presented only one ureteral orifice, and its walls were thinner than usual. The penis, urethra, and testicles were normal, and the left

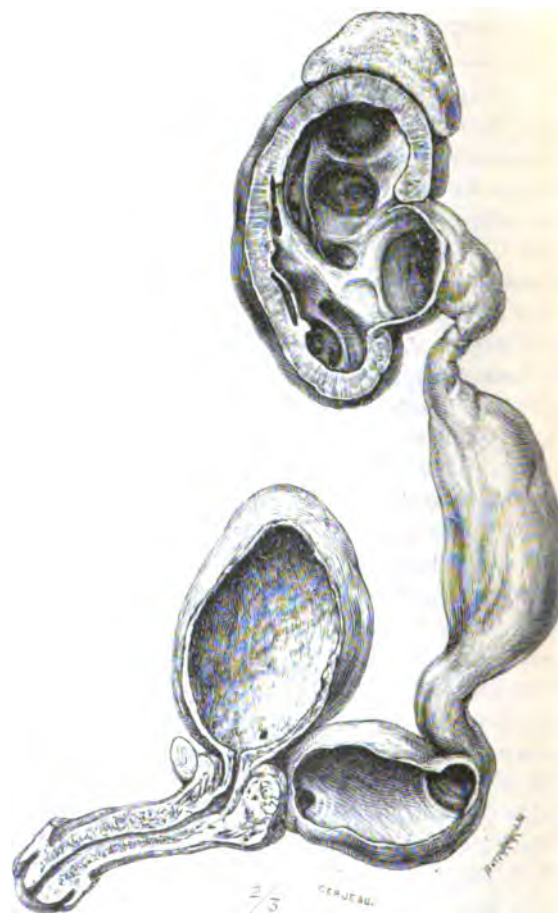


Fig. 2.—The urinary organs with the right adrenal of a new-born child.

adrenal occupied its usual position. No traces of the left renal artery, vein, or ureter were found. The anus was normal.

In this case dissection failed to bring to light anything to account for the distension of the excretory ducts of the kidney, but it clearly indicated that mechanical obstruction of some kind

interfered with the flow of urine through the vesical orifice of the ureter.

CASE 4. *Torsion of the penis in an infant.*—During 1896 a baby boy, three months old, was brought to me by his mother because the penis, instead of hanging downwards, was directed laterally towards the left thigh, and when he voided urine there was difficulty in directing the stream into a convenient receptacle.

On examining the parts it was easy to observe the twist in the penis, and when the organ was brought into a natural position, on being released it at once resumed its abnormal deflection to the left.

This penis, as is the case with twisted penes in general, is unusually large, and it lacked a frænum; there was also a groove on the under surface of the

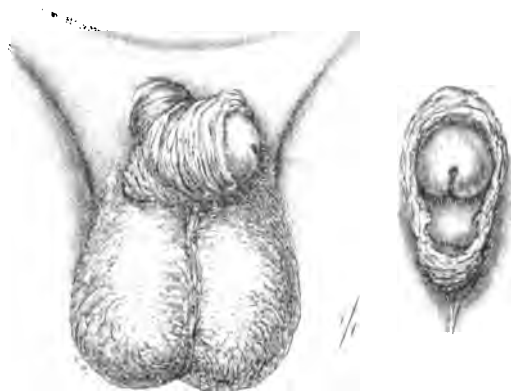


Fig. 3.—A twisted penis. The small figure shows the groove on the glans and the absence of the frænum. (Nat. size.)

glans representing a minimum degree of hypospadias. At the angle of torsion there is a sac-like pouch of skin resembling a pressure bursa. I did not attempt or advise any form of treatment, and regret that I have for some months lost sight of the case.

Torsion of the penis is a condition of some interest. I am of opinion that it is due to the penis being squeezed between the pelvis and the thigh when the lower limbs are acutely flexed on the trunk during intra-uterine life; in this instance the existence of a pressure bursa at the angle of torsion is strong evidence in support of this opinion. The condition also throws light on the mode of origin of some examples of pre-natal hydronephrosis. It is certain that the kidneys

excrete urine in the late stage of foetal life, and many specimens of congenital hydronephrosis, in some of which the ureters and renal pelvis were converted into large sacs, and the muscular fasciculi of the bladder exhibited true hypertrophy, indicating that the seat of obstruction was anterior to the urethral orifice of the urinary bladder; yet on careful examination nothing was found to account for the retention. It has occurred to me that compression of the penis between the thighs, or between the thigh and pelvis when the legs are flexed on the trunk during foetal life, might explain the retention.

Whether this be true or not, it is an undoubted fact that torsion of the penis and bilateral hydronephrosis are sometimes associated. In the present case I very carefully palpated the loins but failed to detect any evidence of sacculated kidneys.

The slight hypospadias is of interest as an addition to the evidence in support of the view that epispadias is really hypospadias occurring in a twisted penis.

SOME PRACTICAL HINTS

ON THE USE OF

PESSARIES IN FLEXIONS AND PROLAPSE.

BY

J. A. MANSELL MOULLIN, M.A., M.B., &c.,

Physician for Diseases of Women, West London Hospital;
Physician to the Hospital for Women.

GENTLEMEN,—I feel I am somewhat at a disadvantage in giving you a lecture after the interesting discourses you have heard from my colleagues. They were able to exhibit to you patients and specimens and magic lantern illustrations, and in other ways to excite your attention and interest. The nature of my subject prevents my bringing before you anything but these pessaries and a few diagrams. I shall, however, endeavour to make my remarks as practical as possible, and may perhaps be able to give you a few useful hints.

Before commencing I may say that no amount

of lectures or reading will make a man proficient in the use of these instruments. It is only by constant practice on a large number of patients that such proficiency can be attained, and a knowledge acquired of the instrument most suited to any particular case. Nowhere can that knowledge and practice be acquired better than in the out-patient department.

It is not my intention to give a description of all the pessaries which have been invented. Their number would fill an instrument maker's catalogue. I am only going to speak of the commoner instruments, such as are required in every day gynæcological practice. There are certain rules which are applicable to the use of pessaries of all kinds.

(1) Be sure of the diagnosis. It is not always so easy to differentiate a rounded tumour behind the uterus from the retroflexed body of that organ. Although the uterine sound in most cases will determine the point, yet such difficulties do occur.

(2) Ascertain that the uterus can be restored to its normal position without difficulty or pain.

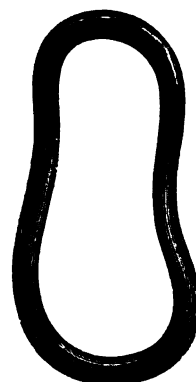
(3) Be sure the pessary fits easily, without pressure on bony parts or causing distension of the vagina.

(4) Instruct patients how to remove pessaries and to re-insert them. I direct all pessaries to be removed at night. An exception to this rule may be made in the case of a Hodge pessary, which can only be replaced satisfactorily by the practitioner.

(5) The vagina should be douched with Condy or some antiseptic on removing the pessary for purposes of cleanliness.

The Hodge pessary is the one which claims our consideration first. I have several varieties here, alike in shape, but different in material—gutta percha, white metal, vulcanite, xylonite, etc. The first two have the advantage that they can be readily moulded at the time of use to the shape desired. A few words on the action of this pessary. It appears to me absurd that the antiquated idea of the lever action of this pessary should be retained in modern text-books, but such is the case, and we find it still named the Hodge or lever pessary. It is certainly nothing of the kind. The Hodge pessary is simply a "splint" or "chair," and nothing else; a mechanical contrivance for supporting the uterus, just as a limb that has been broken or displaced is supported by

a splint. What we have to learn is how to select a suitable splint, and how to apply it.



Now a splint is of no use unless it supports both ends of a fractured limb. So with a Hodge pessary, unless it rises sufficiently high to support the fundus uteri it is of no use. In other words, the posterior vaginal *cul de sac*, or fornix, must be sufficiently deep to allow the pessary to rise behind the fundus, or the body of the uterus will simply retain its faulty position and fall over the top of the pessary.



When the cervix uteri is inserted into the apex of the vagina a Hodge pessary is useless, and should it be necessary to correct the position of the uterus some other means must be employed. The insertion of a retroflexed or retroverted uterus into the apex of a short vagina is a congenital condition which from time to time comes under our notice, but does not *per se* call for treatment. Secondly, a splint to be effective must be steady. The vagina is an elongated cavity, the walls of which are in apposition. The outlet or floor of the vagina is constricted by a sphincter, a thick, elastic, muscular structure. Any pessary or other body placed within the vagina remains there passively unless the outlet has been so damaged that it falls away by its own weight.

The pessary selected must be adapted to the shape of the vagina, and fill it without distending it. It is useless placing a small pessary in a large yielding vagina, where it only rolls about and fails to exercise its action. When the vagina, therefore, has lost its original shape and tone, and becomes a mere flaccid cavity, we must turn to other means. We then have a valuable resource in the round watch-spring ring pessary, which acts upon a somewhat different principle. A Hodge pessary should be worn continuously, and does not require to be removed at night. It does not interfere with intercourse, and conception often takes place while it is being worn. Indeed, it is not uncommon for a patient who has been sterile for some years to conceive at once as soon as the position of the uterus is corrected.

As the pessary requires accurate adjustment, and there is some difficulty in placing it properly behind the cervix, the patient cannot be trusted to replace it when removed. It should be attended to at stated intervals of six to eight weeks.

After a time, in a case where the use of a pessary is clearly indicated, the engorgement subsides, the tenderness of the fundus disappears, and the inevitable dragging and bearing down are relieved. Nothing is more certain than the benefit afforded by a well-adjusted pessary. I am doubtful, however, if a permanent cure is ever effected, or the natural supports of the uterus ever regain their tone. Once the abnormal position is acquired, the tendency is to return to it; and even though pregnancies intervene, after labour the uterus inevitably returns to its former condition.

Nothing has led to more erroneous practice than the idea that this pessary acts as lever. We find it stated that when adhesions bind down the retroverted body of the uterus the lever pessary will still be useful. The adhesion will slowly stretch under the continuous gradual action of the lever, and under the gradual elongation disappear by atrophy. Now, when the uterus is at all bound down by adhesions, or difficulty is experienced in replacing it either by the finger or the uterine sound, a matter which can readily be determined by a practised hand, no pessary is permissible.

Rule 2 is most explicit on this point. The gravest consequences may follow neglect of this rule. It is obvious that any adhesions which bind down the uterus are the result of previous attacks

of inflammation or peritonitis; and the possibility, or rather probability, of starting this afresh must not be lost sight of.

As a matter of fact, the fundus of the uterus is seldom simply bound down by adhesions attached to itself. When the uterus is found to be fixed and immoveable it is nearly always due to primary disease and inflammation of the appendages, which, by contracting, draw the fundus uteri backwards and fix it in that position.

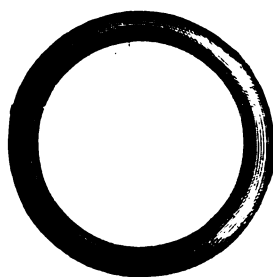
The insertion of a pessary under such conditions, if it has no worse result, generally causes so much pain that the patient finds it necessary to remove it. So far from the uterus giving way and rising in the pelvis under the influence of the pessary, it only presses on the latter and tends to drive it down. Twice just recently I have found it necessary to open the abdomen in cases of this kind. In both the ovaries and tubes were diseased, and firmly adherent to the back of the uterus. The fundus and sigmoid flexure of the colon were also inseparably connected in a mass of adventitious tissue, but having succeeded in removing the appendages I deemed the condition of the fundus a matter of small importance.

One more point to which I wish to draw attention. A retroflexed uterus may be permanently flexed upon itself, or the uterine walls at the junction of the body with the neck may be so weak and flaccid that the body may assume any position either forwards or backwards. The explanation of this is not very clear. Possibly a uterus which has acquired a retroflexed form at the time of puberty, and may be said to be naturally retroflexed, is less capable of movement than when the flexion is the result of childbirth or some pressure acting upon an enlarged, flaccid, and heavy uterus. When the uterus is permanently flexed on itself the application of a pessary is not very successful. When the fundus, on the other hand, is freely moveable, a well-fitting Hodge pessary, by restoring the shape of the vagina and drawing it upwards and backwards, draws the cervix in the same direction and tilts the fundus forwards.

I have already mentioned the watch-spring ring pessary in cases where the vagina has lost its tone and shape, and is unable to retain a Hodge pessary.

The ring pessary must be of a suitable size,

sufficiently thick, and the spring sufficiently strong to retain its circular shape. It should always be removed at night and replaced in the morning by the patient. The action of the ring differs from the Hodge in that it gets no support from the vagina, but finds its resting point on the walls of the pelvis, the spine and tuberosities of the ischium,



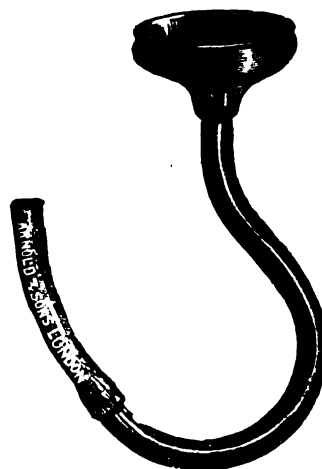
and the sacro-sciatic ligaments extending backwards from them. Its action is that of the Hodge above described. By drawing the vagina backward it draws the cervix in the same direction, and so tilts the fundus forwards. It does not materially raise the uterus, but it prevents further descent, and is a very useful pessary in practice. I often use it in preference to a Hodge when it fits the vagina comfortably. It is quite unsuitable where the vagina retains its shape, not having been distended by childbirth or some other cause. In cases of complete prolapse of the uterus, ring pessaries are seldom sufficient. The invagination of the vagina and the pressure from above almost invariably force out the ring and uterus together. The ordinary sizes of the ring pessary used are $2\frac{1}{2}$, $2\frac{3}{4}$, and 3 inches in diameter. A ring above this size frequently causes inconvenience by pressing on the rectum.

In complete prolapse Zwanck's pessary will be found more efficacious. There are endless varieties of this pessary, but the one I have here is certainly the best. It takes its rest upon the same part of the bony pelvis as the ring pessary, and like it is only suitable when the vagina has been dilated and lost its original shape and tone. No Zwanck's pessary, however small, can be inserted into a nulliparous vagina. It should always be removed at night. The patient after a little practice will have no difficulty in managing it for herself. It should be freely moveable in the pelvis, not press-

ing on the bony walls, or causing tension of the vagina. The sizes most frequently required are

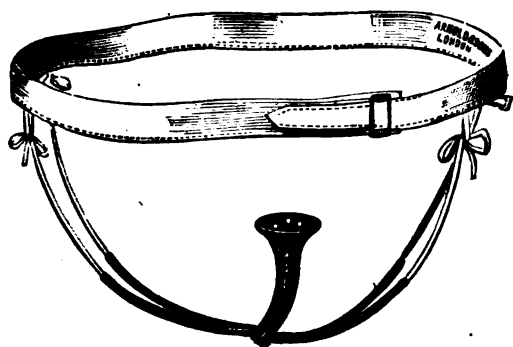


3, $3\frac{1}{4}$, and $3\frac{1}{2}$ inches. In the event of the pelvic walls affording no resting-place for the wings of the pessary, we must have recourse to the cup and stem pessary, or some modification of Cutter's pessary which obtains its support from the waist-belt. No little trouble is often expe-



rienced in supporting the prolapse of elderly women when the vagina has become atrophied, and will not allow the introduction of a pessary sufficiently large to rest upon the pelvic wall. I do not think I need take up your time with a discussion on prolapse. For all practical pur-

poses it is sufficient to know that retroflexion and retroversion are the early stages of descent of the uterus, and that if it continues to progress the appearance of the uterus outside the vulva is only a matter of time. In descent of the pelvic floor, the whole or a part only may be affected. In



some cases the anterior vaginal wall and bladder are the first to appear outside the vulva (cystocele), then the cervix, and finally the posterior vaginal wall. My own experience, however, is that the cervix is generally the first part to appear at the vulvar outlet. The posterior wall in its lower part (last inch) is closely adherent to the perinæal body, and rarely completely descends.

You will not be likely to mistake hypertrophic elongation of the cervix for prolapse of the uterus.

I have endeavoured to give you as briefly as possible my own views on the use of pessaries. I should add I do not make the same extensive use of pessaries for minor degrees of displacement that I formerly did. Pessaries are undoubtedly very objectionable instruments for many reasons; and when we consider how very little suffering is sometimes caused by prolapse in the major degree, a suspicion arises that the importance attached to the minor forms is somewhat overestimated. Where the indications are clear the best results attend their use, but it does not necessarily follow because a uterus is retroflexed that the symptoms complained of are due to that fact. Neither is it possible with any pessary devised to restore every case of flexion and maintain it in position. The less we talk to our patients about "misplacement" the better. It is a still greater mistake to insert a pessary unnecessarily. The female mind becomes so impressed with the idea of the necessity for the artificial support,

whether it does any good or not, that the subsequent conduct of the case becomes a matter of real difficulty.

I must add I remove almost as many pessaries as I insert; and on a first visit make a point of removing a pessary to watch the effects on the next occasion. I find many have been inserted without the smallest pretence of reason.

A lecture of this kind would probably be considered incomplete without a brief reference to the operative measures available in these cases. We are passing through an era of gynecology which may appropriately be termed an "operative era." There is a danger that the interest excited by the brilliant results of modern abdominal surgery may cause us to lose sight of and underrate the value of some of our former means of treatment. But there is ample scope for both, the more recent operative work and what there is of value in our old methods of treatment. I would point out, moreover, that very many recognised gynecological operations have fallen into disuse, and very many of the more recent operations are still waiting to have their value proved.

Shortening the round ligaments (Alexander's operation) is the one which perhaps has aroused most attention. The round ligaments are exposed on either side by an incision upwards and outwards from the pubic spine, in the direction of the inguinal canal. The incision is about two inches in length, depending upon the amount of fat which covers the parietes. When found the ligaments are separated from their connections in the inguinal canal and gently drawn forwards. They are then secured to the pillars of the external abdominal ring by two or three fine buried sutures. Excellent results are claimed by Alexander and one or two others for this operation. Its disadvantages are that the ligaments are often very difficult to find, and this difficulty necessitates a longer incision. The tendency to hernia is then increased. It is a necessary condition, moreover, that the uterus should be moveable. This latter appears to be its most serious defect. It is just in those cases where the uterus is free from adhesions and freely moveable that a Hodge pessary answers all requirements. There seems, then, to be really only a limited scope for the operation, viz. in those cases where for some reason, there being no complications, a Hodge pessary fails to act.

In prolapse I cannot see that shortening the round ligaments can be of any use. The normal structures having given way, either from debility or the strain imposed upon them, the whole pelvic floor descends, and it is impossible to conceive that all this amount of mischief can be rectified by two unimportant ligaments, attached by two or three fine sutures to the external abdominal ring.

Dr. Abbe (New York) says that when the ligament has been drawn out and cut off at the external abdominal ring, it is hopeless to expect it permanently to seal itself in the elastic tissue of the new inguinal scar with the uterus dragging constantly upon it. He therefore utilises the round ligament itself for the double purpose of sewing up the inguinal canal, and securing the ligament immovably.

"Primary union invariably occurs after the operation, and in thin subjects the buried natural suture can be felt for weeks. The results are all that can be desired." I have no personal experience of this plan, but feel somewhat sceptical that a self-respecting ligament would allow itself to be treated in such a way.

Hysteropexy, or Ventral Fixation.—This operation is less scientific than the preceding, but it possibly has some advantages. The peritoneum may be opened or otherwise, the condition of the uterus and appendages ascertained if necessary, and the fundus uteri attached by means of sutures to the linea alba. It appears to me that, like Alexander's operation, it might have a limited scope in cases of retroflexion which cannot be otherwise treated, but that it must be useless in prolapse.

The adhesions so formed would probably soon stretch, and buried sutures are never to be depended upon.

Vaginal Fixation.—In this proceeding the peritoneal pouch between the uterus and bladder is opened up, as in the anterior incision for vaginal hysterectomy. The retroverted uterus is anteverted into the wound, fixed there, and the vaginal flaps closed over it. This is the so-called vaginal fixation of the uterus.

Apart from the immediate result of these operations it is important, when they are performed during the child-bearing period, to ascertain what consequences would be should pregnancy

occur. According to Dr. Noble (Philadelphia) the results are simply disastrous. In one fourth of the cases in which pregnancy occurred abortion was the result, and in those which went to full time serious obstetrical complications arose, necessitating delivery by forceps, version, craniotomy, and even Cæsarean section. He regarded it, therefore, as unjustifiable during the age of child-bearing.

Vaginal extirpation of the prolapsed uterus is advised by some, and has been carried out frequently in America and on the Continent. It is not practised in this country. I should doubt if it cured the prolapse altogether, and I think would further weaken the pelvic floor.

Porro's Operation.—A case occurred to me quite recently in which an elderly woman, with both ovaries cystic and distending the abdomen, for which the operation was undertaken, suffered also from prolapse of considerable size. Instead of ligating the pedicles and returning them, I drew up the uterus, which was atrophied, and included the whole in the wire loop, fixing it outside the parietes. I shall follow the case with interest to see the ultimate result. She will have six weeks in the recumbent position, in which the prolapsed organs may regain their tone, if they will, and there will be no question as to the union of the stump with the abdominal parietes.

I have another case in hand, in which the patient is only 42 years of age, the uterus enlarged and filling the pelvis. A Zwanck's pessary supports the prolapse. I believe removal of the uterus will afford this patient much relief. In such a case Porro's operation appears to me to have decided advantages over vaginal extirpation, and in suitable cases to be a decidedly good proceeding.

Hepatic Complications of Typhoid Fever.
—Dr. Osler considers this a rare complication, and when it occurs is apt to be mistaken for appendicitis. He reported a case which had been operated upon for appendicitis, and on opening the abdomen the appendix was found to be normal. The gall-bladder was much enlarged and inflamed, and contained a quantity of fluid, which was withdrawn, and the colon bacillus found to be present. An operation for the removal of a gall-stone was performed some time afterwards.

Pediatrics, June 1st, 1897.

DEMONSTRATION OF CASES AT CHARING CROSS HOSPITAL,

By STANLEY BOYD, M.B.Lond., F.R.C.S.,
Surgeon to the Hospital.

Specimen of Cancer of Breast (from the case reported in CLINICAL JOURNAL, June 2nd).

GENTLEMEN,—You will remember that when we last went round the wards I showed you an extremely bad and rapid case of cancer of the breast, with great swelling of the arm and side of the trunk. I then considered the advisability of employing the recently suggested treatment of inoperable mammary cancer by double oöphorectomy; but I finally decided that the disease was in this case too far advanced to allow any reasonable hope of success. At that time the patient was suffering from dyspnoea, due, as I believed, to fluid in the left pleura. To relieve her, we drew off thirty ounces of fluid, which contained no trace of blood. The fluid returned rapidly, and we endeavoured to tap again; but although I put the whole length of an aspirator needle into the chest wall, I could not reach the fluid, so thick was the wall from oedema. The patient gradually sank from exhaustion and dyspnoea, and died in two to three weeks. I now have the breast to show you. You will remember that the skin was becoming vascular and infiltrated with extreme rapidity. There was hardly any discoloration on admission, but later we noticed it spreading every day, from the left breast across the mid-line. You can now see the cancerous infiltration of the skin with the naked eye; it is from a third to half an inch thick. There was one mass of cancer running thence right through the breast, infiltrating both pectorals, and adhering directly to the chest wall. Here the solid mass of cancer broke up, and there were discrete nodules in the intercostal muscles. On opening the chest and looking into the pleura, which was full of fluid, there were many nodules on the external parietal pleural surface. The apex of the left lung was adherent to the pleura, and on the lung side of this adhesion there was a small mass of cancer as large as the end of a thumb. There were no other secondary visceral growths found. The left lung was absolutely collapsed,

and weighed $9\frac{1}{2}$ oz. The right weighed 24 oz., showing what a lot of blood and fluid is contained in the ordinary tissues. Next as to the cause of the swelling of the arm—of the so-called lymphoedema, a name which might seem to imply that it was due to obstruction in the lymph current rather than in the veins. But we all know that obstruction sufficient to cause oedema is much more common in veins than in lymphatics. If the lymphatic glands on one side of the neck be completely removed, as far as we can, oedema does not occur; indeed, a very free removal may be practised upon both sides without ill result. The only instance I can remember in which I have been led to suppose that the removal of lymphatic glands led to oedema was a case of very early cancer, in which I removed the breast very freely, together with all the axillary glands I could find—none being obviously diseased. That woman developed oedema of the arm, and although I was unable to prove it by excluding venous thrombosis, it did seem likely that this was a case in which removal of a large number of acting glands resulted in oedema. When we remove a lot of tubercular glands, the reason why untoward consequences do not, as a rule, arise is that their removal has really been gradual and due to the disease, so that when they are taken from the body, they are not active glands, and a collateral lymph circulation has presumably been established. But, in the case I have mentioned above, I removed apparently healthy glands suddenly, and a long persistent oedema of the arm resulted. In the case from which the specimen which I am showing you was taken, I dissected out all the vessels up to the collar bone. The axillary artery high up was very considerably constricted in the cancer tissue. I slit up the veins, and at three points between the lower part of the axilla and the junction of the subclavian and jugular veins there were rounded cancer masses growing into the axillary vein, greatly narrowing the channel, but still covered with epithelium. Three obstructions of this character would obviously oppose a great difficulty to the return of blood, and would alone, as I think form a sufficient cause of the lymphoedema; though, doubtless, innumerable lymphatics were blocked besides. Probably the swelling would have been still more extreme if the artery also had not been constricted.

Specimen of Fibro-myoma Uteri.

I also wish to show you here a tumour removed from a case which we will look at upstairs. You see it is a uterus as large as if six months pregnant; it contains a large, soft, sessile fibroid, which grew from the fundus and anterior wall into its cavity. The woman was blanched by repeated hæmorrhage; no medical treatment had long benefited her, and hysterectomy was indicated, which I performed. The specimen is much smaller than when I removed it. In the first place enough fluid came from it to fill a soup plate, and it has been left in formalin ever since, which constricts the tissues.

Ossification of Tendon of Adductor Longus.

I want you to just look at this woman, æt. 59. She was knocked down by a hansom cab on December 14th. She does not know how she fell, but the principal injury was to the right ankle, and she was bruised in various parts. Two weeks after she was in hospital she discovered that she had a swelling about the pubes, and she now says that it hurts her when she walks, and when she moves the muscles of the leg. On examination you will find that there is a pointed, bony swelling one and a half inches long, which starts from the pubic bone, just on the right of the mid-line. The tendon of the adductor longus stands out clearly when I make her contract the muscle, and the swelling lies in the tendon of origin of the adductor. It is apparently an ossification in this tendon, although on the other side there is no indication of anything of the kind, and tendons usually ossify symmetrically. What is the reason she is suffering pain from it now? We are all familiar with the cases in which, when persons become aware that they have got something the matter with them, they find also that it becomes painful. Sir James Paget tells of a lady who had a cancer of the breast, which she had discovered a few days before she went to see him. In reply to his question, "Is it painful?" she replied, "Only since I noticed it." The only other solution of this present case would be that the woman had sustained a fracture of the pelvis of a serious nature, with displacement of fragments, and that it was not discovered when she was in hospital and had no need to walk about. But you will be able to feel that the upper rim of the

pelvis shows no trace of deformity or of callus, and the pubic arch immediately below the bony spine appears to be normal. So I think we may safely regard it as a case of ossification in the adductor longus tendon on one side only.

A Submaxillary Nævus with misleading History.

This woman, æt. 38, has a swelling below her jaw. The history is that for twenty-four years she has noticed a swelling in the left submaxillary region. When she came in we thought it was about the size of a large walnut. She says that with tolerable regularity it increases in size for two or three days, and then diminishes, the diminution being accompanied by a sort of bursting and discharge into the mouth, which she compares to saliva. The discharge is so free that she has to use four or five handkerchiefs to spit into. About once in every two or three months the discharge is yellowish and tastes bad, but ordinarily it is just like saliva. This discharge, she thinks, enters the mouth far back under the tongue. When the swelling reaches its fullest point there is some little pain, then the discharge takes place and everything quiets down. We found, on her admission, that the swelling was very soft, slightly lobulated on the surface, and that it could be pushed about somewhat beneath the skin, when its lobulation became visible. The skin was normal, except that at two spots it had a thin, bluish, translucent look. We did not notice any swelling up after pressure like a nævus. She was not long under observation, but one observer at least thought he had noted diminution in size. The whole history was so complete that the diagnosis of a cyst in connection with the submaxillary gland seemed an obvious one. We could not find any abnormal opening into the mouth, and supposed that the discharge occurred through Wharton's duct. I proposed to cut down from the outside upon the cyst, dissect it out, endeavour to find the opening in the mouth and close it. My surprise was very considerable when, at my first incision, I found that I had to deal with an ordinary cavernous nævus, occupying the whole submaxillary region superficial to the salivary gland, which was quite normal. By the sacrifice of a little blood I got all the way round it and removed it.

Nævous tissue will always bleed if you cut *into* it; oozing is then continuous and rapid. Therefore, in removing a nævus always keep *outside* in the healthy tissues around it. I show you the nævus which was taken away that you may note its sponge-like, trabecular structure, and be able to recognise it in the living subject.

The patient has had no discharge from the mouth since the operation. I do not know what "suggested" this discharge to her, but it would seem to have had a subjective cause. This case shows how much we may sometimes be misled by a history, and tends to breed scepticism with regard to patients' statements; yet every now and then, on the other hand, we have to regret not having paid more attention to a particular history.

Tubercular Elbow; Excision.

This little girl, æt. 14, had a history of pain and swelling about the elbow for ten months before coming in here last February. She was treated by incomplete rest and iodine painting. Just before Christmas, 1896, an abscess burst on the inner side of the elbow above the epicondyle, and since then another has burst. After Christmas a swelling formed in the axilla, and increased rapidly. When she was admitted we found her elbow diffusely and greatly swollen, the swelling extending from three inches above the joint to two inches below it. There were two sinuses just above the inner condyle, opening in the line of the scar left by incision of the abscess which first formed. On the outer side between the olecranon and the condyle there was a red fluctuating area, and the skin was tense and shiny. Pressure here caused oozing of pus from the internal sinuses. The arm was fixed at an angle of about 55°, and pronation and supination were reduced to 5°. The axillary glands formed a subpectoral swelling, which was very obvious to the eye. Altogether, therefore, there was a very unpromising state of matters. I immediately opened the abscess on the outer side of the olecranon on February 8th, and on February 13th I did the regular operation of excision of the elbow. First of all I opened up the two sinuses on the inner side. On reaching the humerus I found that there was a large hole leading into it, and that its lower end was much "expanded." I cleaned out the cavity, and then found that there was a way down into the elbow-joint. It was

clearly by this track that the pus which escaped on pressure over the head of the radius had passed through the joint. I opened the joint by the ordinary long median posterior incision, separated the triceps from the olecranon, and then cut off the top of the ulna. I found that the whole of the olecranon process, as I have shown in this drawing, was tubercular, and I had to gouge out the shaft below the coronoid process to make reasonably sure that all disease had been removed. The radius was only superficially affected, and I removed its head with a knife. Lastly, I cut away the whole lower joint surface of the humerus, and through that large hole I looked into the cavity of the expanded shaft and cleared tubercular tissue out of quite the lower fourth of the bone. Nothing but a mere shell was left here, and the part as a whole looked almost too bad to be saved. I treated by plugging the cavity firmly with iodoform gauze,—a somewhat painful, but most useful form of dressing. There is now practically no discharge, and the large wound is now healing up in such an astonishing way that to avoid a stiff elbow we have to employ daily forcible movements. We are further trying to develop the wasted muscles by regular faradism.

The primary source of disease having been removed, the large mass of glands in the axilla has all but gone—very much to my surprise, I must confess. I have never seen such a mass of tubercular glands disappear; and if I had not had a long operation on the elbow I should certainly have removed the axillary glands at the same time.

The explanation probably is that the resistance of the gland tissues was of no avail against the tubercle bacilli so long as fresh supplies of organisms (both tubercular and septic) were constantly being conveyed to them by the lymph-stream; but when this was prevented by the treatment adopted, the tissues were able to struggle successfully against the organisms present in them.

Fibro-myoma Uteri; Hysterectomy.

The next patient I want to show you is a single woman, æt. 34, a servant, whom I mentioned before we came into the ward in connection with the specimen of uterine fibroid which I showed you. She came complaining of losing too much at her periods, the discharge lasting latterly for a fortnight at a time, and being very profuse.

There was no metrorrhagia or loss during the fortnight's interval. The weakness consequent upon this became so great that at last she had to spend much of her time in bed, and she got excessively anæmic. Two years ago she noticed that she began to get stout about the abdomen, and that she was losing flesh elsewhere. No tumour was discovered, however, until she was admitted into this hospital three months ago, on account of uterine hæmorrhage. Dr. Black kept her in bed, and put her on ergot. This arrested the loss, and she went out. But with the next period the bleeding came on as profusely as ever, and it became obvious that something radical must be done to control it. Moreover, the tumour was growing. The state of matters was that she had a perfectly smooth, pear-shaped swelling, which rose above the pubes in the mid-line to some two or three fingers' breadth above the umbilicus. It was a little higher on the right side than on the left. The tumour seemed fixed, but it was not certain whether the rigidity of the muscles did not account for that. It was so soft that, in some of the notes, it is described as semi-fluctuating. *Per vaginam* you could reach the cervix high up, drawn up by enlargement of the fundus—so much so that the anterior lip had disappeared. Posteriorly there was still a small fornix remaining. The cervix was normal, and there was no growth starting from it. This we were, of course, glad to find, for in cases of hysterectomy in which the broad ligament is opened up by the tumour there is very free bleeding, which you cannot see to stop until you have got the tumour out, and a great deal of blood may be lost before this is effected. We next found that there were no bladder or rectal symptoms; the urine was normal, and there was nothing to indicate that there was pressure on the ureters. The sound passed four to six inches, apparently into the centre of the mass, leading to the diagnosis (faulty, as you have seen) of diffuse fibroid thickening of the uterus.

On March 13th I did an abdominal hysterectomy, and there was nothing unusual about the operation. I had to make a long incision at once from above the umbilicus down to the pubis. The umbilicus was too deep for thorough disinfection, so I removed it entirely. Through this incision I could just squeeze the tumour. The recti remained rigid in spite of the anæsthetist's

efforts, and rendered the operation more difficult by contracting the space I had to work in. As the tumour came out I saw a number of large veins which came off from the right of the uterus in the direction of the ovarian artery. They were tied in two places and cut between, and that let me into the broad ligament. I divided the peritoneum until I had reached and tied the uterine artery on the same side. On the left side the ovary lay high, and was closely bound to the psoas, so that with the rigid muscles I could not tie the ovarian plexus outside it. I applied two ligatures to the veins coming from the uterus just inside the ovary, and divided the broad ligament here. I intended to go back for that ovary, but I did not do so, and it may turn out to have been a good omission. I now got down on the left side of the large tumour and secured the left uterine artery. Lastly, I raised anterior and posterior flaps, chiefly of peritoneum, and removed the uterus by cutting across at about the level of the internal os. Very little blood was lost. All the vessels were tied, and then I sewed up from one ovarian pedicle to the other across the stump of the uterus. The flaps were brought together by deep stitches, and the peritoneum was sewn up again, so that there was a transverse line of stitching right across the pelvis. Everything was restored to its proper place, and the abdominal wall was closed by three layers of stitches. The patient has done most satisfactorily, in spite of the fact that she had a very foul vaginal discharge, which was clearly a source of danger.

Later on two or three slight rises of temperature caused me to make an examination, when I found that the stump of the uterus was fixed, and that a considerable exudation had formed, chiefly posterior to it. Under rest and douches this subsided, and she left the hospital greatly improved in every way.

Idiocy ; Linear Craniotomy.

On this idiotic child, 2½ years old, I have performed the operation of linear craniotomy. When brought in he had double optic atrophy and was believed to be blind; he apparently heard; he kept his head retracted rigidly, his limbs rigidly flexed; the thumbs always remained in the palms with the fingers closed upon them. There were no clonic spasms. The head was not

strikingly small, but the facies of a low type of idiot was well marked. He lay just as he was placed, cried a great deal without obvious reason, and swallowed with extreme slowness.

The operation is a very simple one, and has for its object to render the skull yielding and to allow the brain to grow if it will. I thought that this idea could be best carried out by setting free the top of the skull. Accordingly on two occasions (a fortnight intervening), and with every precaution against shock, I turned down one or other side of the scalp from near the mid-line and excised a strip of bone half an inch wide, together with its periosteum, from the side of the skull. The two cuts were connected by a cross cut anteriorly and nearly connected posteriorly. The brain was thus covered by a sort of valve of bone easily depressed by finger pressure. With Keene-Hoffman forceps the operations took twenty to twenty-five minutes, and shock was quite moderate. The wounds healed well. I am not in a position to express any opinion on the question whether the brain fails to grow and thus leads to early synostosis of the bones, or whether the premature synostosis is primary and prevents the brain from growing; but the skull was irregularly and greatly thickened in this case, and undoubtedly abnormal elsewhere than at suture-lines. So far, the results of the operation have been very slight. The nurses report that the child cries much less, and that he does not take quite so long in swallowing. The rigid flexion of the limbs has given place to rigid extension, with marked adductor spasm of the thighs. (A month later no improvement had occurred, and the tendency to flexion of the limbs was again marked. The bone flap had again become fixed by ossification across one at least of the grooves cut in the skull.)

NOTES.

The Treatment of Constipation in Infants.

—The 'Journal des Praticiens' of January 9th, 1897, contains a practical article upon this subject. First, it deals with the local accidents which may produce constipation, calling to mind the fact that purgatives should not be given until we

are confident that umbilical or other herniæ do not exist. It may be, too, that prolapse of the rectum or an anal fissure may be a factor to be considered. It is not to be forgotten, also, that fever and cutaneous eruptions sometimes arise in children as the result of constipation. An important factor to be considered in treating these cases is that of heredity and conformation of the intestine. Children of gouty parents frequently suffer from atonic bowels, and in other cases the intestines and abdominal wall seem to be so relaxed as to predispose to this condition. The employment of sterilised milk also favours constipation, and the administration of farinaceous articles too early in life, by provoking dyspeptic troubles, may either result in diarrhoea or constipation. The question of modifying the diet of children, therefore, is of very great importance. If old enough to receive vegetable substances they should be given Graham bread, which leaves a large residue; the ordinary vegetables, such as string-beans and peas, and from time to time mild laxative substances such as manna or cascara, should be given. Frequent exercise in the open air is also a necessity. Very frequently adding a little sugar to the milk, if the child is fed on sterilised milk, will prevent it being so constipating in its effects. Massage of the abdominal area gently applied for a number of minutes morning and night, the skin being rendered oily by the use of vaselin, is also a method which is not to be forgotten. During the massage the fingers should knead the intestines as much as possible. Castor oil and magnesia, while active in moving the bowels, tend to produce constipation to a greater degree after their effects have passed off, although the author of this article believes that calcined magnesia is a useful substance to overcome dyspepsia, and to move the bowel in certain cases. In other instances suppositories and rectal injections produce the best results, particularly suppositories that are made of glycerin. The quantity of liquid which should be used as an injection varies, but ordinarily one or two ounces is sufficient in young children; and if the bowel is not active cold water may be used in place of warm water, and the action of the injection may be increased by the addition of two to three dessertspoonfuls of oil of sweet almonds.—*Therapeutic Gazette*, June 15th, 1897.

TRACHEAL INJECTIONS IN THE TREATMENT OF LARYNGEAL AND PULMONARY INFLAMMATIONS.

By J. A. THOMPSON, M.D.

THE first physician who left us a record of a systematic study of intra-tracheal medication was Dr. Horace Green of New York. Long before the invention of the laryngoscope he had attained remarkable dexterity in passing a sponge probang, saturated with a solution of nitrate of silver, into either bronchus. His reports of his methods and results were so extraordinary that the New York Academy of Medicine appointed an investigating committee to determine the truth or falsity of his statements. The committee agreed that he could and did employ the method described, but a majority condemned its use. With the invention of the laryngoscope there was no immediate revival of the method of direct medication in the treatment of laryngeal and pulmonary inflammations. It is only within the last few years that occasional reference to this subject can be found in medical literature.

There are several reasons for the slow growth of this manner of treatment in professional favour. The principal one is that few physicians are sufficiently expert in the examination and treatment of the upper air-passages to employ it. As a rule they consider all lesions of the nose and throat as local, with but little influence on the health of the patient. This lack of appreciation of the importance of these conditions is largely responsible for the neglect of direct medication. Another reason for the limited use of intra-tracheal injections is the mistaken opinion that they are necessarily painful and irritating. Our ideas of the sensibility of the tracheal and bronchial mucous membrane have been erroneously deduced from that of the larynx. Below the glottis the nerves of sensation are few, and the membrane is not irritated by solutions much stronger than those we use in the larynx. While these reasons have been potent in preventing the more general knowledge and use of this method, there are other and much better reasons for the general use of direct medication in inflammatory diseases of the larynx, trachea, bronchi, and lungs.

By the method of intra-tracheal injection we get

the direct local action of the medicines on the diseased areas. In bronchiectasis no medicine given by the mouth will prevent the decomposition of the secretions in the dilated bronchi. The odour and the absorption of septic material from them cannot be controlled. A few tracheal injections will usually disinfect the cavities, so that the odour disappears and the temperature becomes normal.

It is possible by intra-tracheal medication to produce a rapid and prolonged general effect. Anæsthesia gives daily and hourly evidence of the rapidity with which volatile medicines are absorbed through the lungs, and their effect on the whole organism. From three to five minutes is a sufficient time for an expert to obtain chloroform anæsthesia. You can produce as rapid an effect, and one much more durable, by injecting into the trachea medicines which volatilise slowly at the temperature of the body. Where a dose of menthol had been given in this way, you will find the surface flushed in five minutes; the cold extremities have become warm, and sometimes the patient breaks out in a profuse perspiration. This stimulating action lasts for hours.

Medicines used by tracheal injections are not changed by passing through the digestive organs into unknown compounds. For this reason we can be more certain of their action. No one would think of treating a tubercular laryngitis by internal medication alone. There is just as much reason for applying medicines of known beneficial local action directly to the lungs, as there is for using them in the larynx.

Where medicines are injected directly into the trachea they have no deleterious effect on the organs of digestion. Our valuable expectorants, such as are used in acute and subacute catarrhal diseases, act injuriously on the stomach and intestines. The name of one class, nauseating expectorants, testifies to the universal recognition of this fact. The cure of a bronchitis by direct medication without interfering with nutrition is an advance in therapeutics.

Direct antisepsis can be secured by local medication. It is not possible to obtain this result by remedies given internally. We are prone to forget that in tuberculosis we are dealing with a mixed infection. In the stage of ulceration and breaking down of tissue infiltrated by tubercular matter, there is always a secondary infection by the germs

of suppuration. It is to these, in all probability, that most of the fever, the night sweats, and the other evidences of sepsis are due. We do not attempt to disinfect a leg ulcer by medicating the stomach. It would be just as rational to do so as it is to attempt to disinfect suppurating cavities in the lung by medicine administered *per os*. In cases where tracheal injections can be tolerated, the antiseptic action of the remedies chosen will be very speedily shown, by the subsidence of the cough, by change in the character of the expectoration, and by the decline in the fever.

The administration of medicines by intra-tracheal injection does not interfere with any other line of treatment. Diseases in other organs may be treated or tonics given, while local treatment of the lungs is being used without any incompatibility.

Where medicines are given by the tracheal rather than by the œsophageal route, we can relieve symptoms in hopeless cases without narcotics. We thus avoid their bad effects on nutrition. We also escape their secondary depressing action on the nervous system. A little menthol injected into the trachea will quiet a cough longer and more effectually than will a quarter of a grain of morphia given hypodermically.

Conditions not affected by constitutional medication can be cured by tracheal injections. Gummata in the lung, which resisted all other treatment, have been cured quickly and easily by direct medication of the suppurating cavities.

There are several conditions necessary for success in this method of treatment. The first essential is that the doctor himself be skilful enough in laryngology to do the work rapidly and delicately. The patient must possess a reasonable amount of self-control, and be willing to follow directions implicitly.

It is probable that the medicines used for this treatment should be such as volatilise slowly at the temperature of the body. They should be soluble in the vehicle employed. The solutions used should not be too irritating. The most serviceable solutions are menthol, 2 per cent.; guaiacol, 1 per cent.; creosote, 1 per cent.; camphor, 2 to 3 per cent. Any of these may be combined. In acute diseases the menthol and camphor solutions are most efficient. In tuberculosis menthol and guaiacol give the greatest relief. Guaiacol gives good results in any septic condition in the lungs

or bronchi. The vehicle used should be one of the light petroleum oils or olive oil. Alcohol and water are too irritating, and produce violent coughing. There is ordinarily no spasm and but little cough or distress after an injection of the above solutions. The dose is from one to four drachms. The diseases in which intra-tracheal medication will be of service must be determined by clinical observation.

I wish to present only a few deductions from my experience with this method of treatment during the last five years. The necessary condensation may make my statements seem too dogmatic. All are based on cases actually treated, and are deductions drawn from results thus obtained.

My first notable success was in pulmonary tuberculosis. In August, 1892, I began treating a woman with every symptom of this disease. Her attacks of coughing were so prolonged and violent that she frequently vomited and was unable to sleep at night. An injection of a solution containing menthol 2 per cent., guaiacol 1 per cent. at 4 o'clock in the afternoon would control the cough, so that her dinner would be retained, and she would get a good night's rest. The cough did not return, as a rule, until the following morning. The patient's symptoms entirely disappeared, and treatment was discontinued. A little more than a year after she ceased treatment she became pregnant, and with the digestive disturbances incident to this condition there was a return of the cough, vomiting, and inability to sleep. The treatment by tracheal injection was resumed, again resulting in apparent cure. The pregnancy was uninterrupted, and the patient has had no further occasion for treatment during the last four years.

In other cases of pulmonary tuberculosis, symptoms have been greatly relieved, but I have no other cures where a sufficient interval has elapsed since treatment to make the statistics worth recording.

A few cases that I have seen, where the tubercular disease was making very slow progress, have not been benefited by tracheal injections. Where the diseased process is a chronic, not an acute one, the symptoms have been aggravated by the remedies used.

My next notable success in this treatment was in pulmonary syphilis. Fortunately this is a rare

condition, but there are some cases where gum-mata form in the lung in the tertiary stage, and where the disease is not controlled by constitutional medication. I reported a case of this kind in the 'Medical Record,' October 21st, 1893. I have recently seen this same patient, and there has been no recurrence of the trouble at the site of the original lesion.

In the first stage of acute bronchitis, where there is no secretion, tracheal injections have proved irritating. I have abandoned them at this stage, and give something to induce free sweating instead. In the second stage, after free secretion has been established, intra-tracheal injections of menthol and camphor will relieve the cough more than any other treatment, and daily injections will shorten the attack at least one half, as compared with cases treated by a stomach medication.

In chronic bronchitis there is no comparison between the two methods of treatment. The results are so much more rapid and satisfactory by intra-tracheal medication, that no patient who has once been given this treatment is ever willing to continue internal medication.

There is a condition rarely mentioned, often associated with chronic diseases in the upper air-passages, which gives rise to many of the symptoms that we attribute ordinarily to chronic laryngitis. This is chronic catarrhal inflammation of the mucous membrane of the trachea. You will also frequently find in cases of acute bronchitis that the trachea remains inflamed, as can be seen by laryngoscopic examination, after all the physical signs of bronchitis have disappeared. This is especially true of cases where there is chronic rhinitis and laryngitis, as the underlying cause of the acute attack. This chronic inflammation of the tracheal mucosa gives rise to more or less cough and a feeling of soreness, and sometimes to distinct pain in the chest. Physical examination in this condition shows no signs. It can only be detected by direct examination of the trachea, either by the laryngoscope or by the autoscopic method. Where this tracheal inflammation is coexistent with a chronic laryngitis, the ordinary treatment for laryngitis will fail unless combined with tracheal injections for the cure of the disease of that organ. Stimulant expectorants, given internally, have little influence on the course of a tracheal inflammation. Direct

local medication ordinarily results in a very speedy cure.

Some cases of asthma are notably relieved by tracheal injections. The paroxysm is relieved for hours after the treatment. The bronchitis which usually accompanies this condition is cured more speedily by direct medication than by internal treatment. Some cases of pulmonary emphysema have been given great relief by tracheal injections. The effect was probably obtained through the cure of the chronic bronchitis which usually accompanies this condition.

Where the remedies to be used are not irritating it is not necessary to spray the larynx with cocaine. The injection can be given while the patient takes a slow, full inspiration. The process can be repeated until a sufficient dose has been administered. Where strong solutions or those irritating to the larynx are to be employed, that organ must be anæsthetised. The tip of a properly curved tube of a syringe is then introduced between and below the vocal cords during inspiration, and the whole dose given at once.

To an expert laryngologist there are few technical difficulties in the treatment. There are none which need discourage the family practitioner and prevent him from attempting it. The method is useful in so many pathologic conditions frequently seen by every practitioner, that all should know of it and use it. When they do, they will learn, as a few have already learned, that in the method of intra-tracheal injections we have a means of promptly relieving symptoms, and curing some diseased conditions that are not benefited by any other treatment.—*Journal of the American Medical Association*, June 26th, 1897.

Intermittent Drainage of the Pleura.—R. Macias has secured brilliant results in eight cases of purulent pleurisy by this means, and recommends it for the serous form also. A rubber tube is inserted into the pleural cavity and left permanently, but the free end is closed with a clamp or tied in a knot, and only opened for a while every two hours at first and once a day later. Infection and the entrance of air must be scrupulously prevented, the latter by keeping the free end when open in a vessel containing a weak solution of bichloride of mercury. If the tube becomes obstructed, the clot can be manipulated from without and forced along.—*Rivista Med.*, March 14th

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INTERSTITIAL HEPATITIS.

BY

VAUGHAN HARLEY, M.D.,

Professor of Pathological Chemistry, University College,
London.

THE ordinary term "cirrhosis" of the liver embraces that form of interstitial hepatitis which has advanced to the contracted stage, and although of great pathological and clinical interest, it is at the same time not nearly so important to recognise as the early stages, in which the liver is enlarged, where active treatment may be hoped not only to relieve symptoms but even to produce a cure—if by cure one may call cessation of all symptoms of disease over a number of years.

Before discussing the symptoms and the treatment of interstitial hepatitis, it is as well first of all to shortly summarise the pathological condition present, so that its ætiology may be better understood, and thus the progress of the malady stopped.

The pathological condition consists of a new formation of connective tissue in the substance of the liver, whether around the portal veins or interlobular spaces. It is, in fact, an inflammatory process of the liver, the same as inflammation elsewhere, but not attacking the liver cells proper; the liver cells play no part in this formation, it being a proliferation of connective-tissue cells. There is in the first place an increase in the cell tissue, which later is converted into fibrous tissue. and during this stage the liver is either normal in size or enlarged. In many cases there is, as a result of chill or other exciting causes, an active hyperæmia, which may produce symptoms of its own, such as pain, jaundice, with more or less fever and general discomfort, which at once leads one to suspect the liver to be at fault. However, in the majority of cases when the hyperæmia is little marked, the early stages of the malady show very few symptoms which point direct to the liver,

and therefore the early enlargement of the liver is very often not noticed, and the case may have advanced to late stages before the diagnosis is made.

The new fibrous tissue, as in the case with all scars, by contraction leads to the small hard liver so well known as the "cirrhotic" or "hobnail liver." The fibrous tissue contracting isolates bundles of liver cells, and extending into the acini disturbs the regular arrangement of the hepatic cells, so as to surround smaller or larger groups of cells, and even in some cases to isolate separate cells. This renders the surfaces of the liver either finely granular, or—in the cases where large groups of cells are surrounded—nodular, which may transfigure the organ to such an extent as to make it resemble more a brain with its convolutions than the liver. When the nodules are large they may be felt during life through the abdominal wall, and in some cases by the uninitiated may be mistaken for carcinoma.

The fibrous tissue is rich in blood-vessels, many of which are newly formed. The fibrous tissue, by extending into the nodules of the liver, hinders the nutrition of the liver cells, and hence their atrophy.

In the present article it is not my intention to touch the subject of biliary hypertrophic cirrhosis, but to leave that for a future occasion, and it is specially the condition in the early stages of interstitial hepatitis, where the symptoms do not point so markedly to the liver, that I wish to discuss on the present occasion.

The ætiology of this condition is very various, and that form due to venous congestion from cardiac disease, as well as those conditions of local interstitial hepatitis due to a chronic abscess or an echinococcus, will not be considered here.

The primary causes are grouped by most observers into alcohol, syphilis, and malaria. Various experiments have been done with more or less success to explain how interstitial hepatitis can be produced. In some animals, especially rabbits, after ligation of the bile-duct a more or less definite cirrhosis has been found to occur. However, it

appears that in the older experiments in which this result has been obtained they have been complicated from the operation not having been done with sufficient antiseptic precautions. In the case of man, obstruction to the bile-duct cannot be considered to be a sole cause, since in those obstructions due to carcinoma of the bile-duct or of the head of the pancreas such a condition has not been found present. It is only in cases—and a few of them—in which the obstruction has been due to a gall-stone that interstitial hepatitis has been found in man; and it would thus appear as if bacteria played a rôle in the cause of the interstitial hepatitis, since it has been found that in the case of gall-stones bacteria are often found in the gall-bladder, more especially the *Bacillus coli-commune*; and later observers strongly suspect that the gall-stones themselves are the result of bacterial infection of the gall-bladder.

Other experimenters have tried by ligaturing the hepatic artery, or producing a slow thrombosis of the portal vein, to produce an interstitial hepatitis; but in these the results also have been negative, except in those cases where septic infection has probably been the cause of the results found.

Undoubtedly alcohol is the principal cause of interstitial hepatitis in man. Experimentally, in animals feeding by alcohol has yielded negative results. It has been found in man that wine and beer are not so apt to produce interstitial hepatitis as strong spirits. Formerly beer was supposed to be the principal cause of the large liver with marked fatty changes, but Rolleston and Fenton have lately, in an analysis from a large number of post-mortems, found that, contrary to the usual acceptation, the fatty changes were more marked in the cases of spirit drinkers than in those drinking beer. It is not the occasional drunkard or the person who is supposed to drink who suffers so much from interstitial hepatitis as those persons who consider themselves sober, but are in the habit of continually taking nips between meals; and all cognizant of liver disease at present consider that alcohol, when taken between meals—especially spirits—is the principal cause for the interstitial hepatitis found in man; at the same time, how alcohol produces this condition is an unsettled point. Some consider the alcohol causes a degree of atrophy of the liver cells directly, and

secondary to that the formation of connective tissue; but such a view can receive no support either from experiments or morbid anatomy, and it is very doubtful that if alcohol were even circulating in the portal vein it would produce the condition which we know as interstitial hepatitis. However, the other view, that alcohol produces first gastro-intestinal changes, which secondly lead to interstitial hepatitis, receives strong support from the clinical side, and lately from the experiments on animals made by Dr. Boix of Paris.

The clinical evidence in favour of alcohol producing gastro-intestinal changes are so marked that one can say definitely that scarcely ever a case of alcoholic interstitial hepatitis appears in which one cannot find that during some stage of disease, more or less gastro-intestinal disturbance has been present.

The argument against this view is now very slight since Dr. Boix's experiments, who considered that the gastro-intestinal catarrh would naturally produce butyric, lactic, acetic, valerianic, and propionic acid, as well as higher fat acids, aldehyde, and acetone, and that the absorption of these might lead to an auto-intoxication, and hence the interstitial changes in the liver.

In an elaborate series of experiments he administered these substances to rabbits with their food, and he found in those cases in which he gave butyric, valerianic, or acetic acid, they yielded an interstitial hepatitis, which in some cases resembled the typical cirrhosis of the liver; while lactic acid, the higher fat acids, acetone, and the other substances led to no results. He therefore considered that the products of the gastro-intestinal disease were the real cause of interstitial hepatitis, and alcohol only indirectly played its rôle. That this is probably correct seems to be borne out by the clinical study of cases.

In malaria and syphilis the inflammatory process is produced in each case by its own cause; and in interstitial hepatitis we are dealing with nothing else than a simple inflammation of the liver, which affects the connective tissue. If the liver cells themselves were inflamed, the result would be the formation of abscesses, while in the case where the connective tissue is affected we get in the first place hypertrophy, followed by atrophy. The gastro-intestinal view also explains those forms of interstitial hepatitis which occur in individuals

who have been eating highly seasoned foods, hot curries and condiments, and at the same time have been, strictly speaking, temperate.

The symptoms.—In discussing diseases of the liver it is always customary to talk of jaundice. It may be said at once of interstitial hepatitis, that only in the case when active congestion of the liver accompanies the hepatitis is jaundice ever present, and it is then only temporary, and as a rule not well marked. In these cases urobilin is found present in the urine and *feces*; and as we know now, when no bile reaches the intestine urobilin disappears from the urine. The fact being that in the animal body itself urobilin is not formed, it is only by the reduction of the bile pigments in the intestine by certain reducing bacteria that urobilin is there formed, and it is partially absorbed from the large intestine and to a smaller extent from the lower part of the ileum, to be then excreted by the urine and bile. So that as long as we get urobilin in the urine, although jaundice may be present, we know that some bile is reaching the intestine.

The usual appearance of the patient, instead of jaundice, is merely a slight yellowishness of the conjunctivæ and sometimes the skin. In some cases the sallow colour is due principally to the *anæmia* which accompanies the complaint. The skin is also dry and harsh, and the patient appears not in good health.

Depressed spirits is a very common symptom—in fact, one of the commonest. The mental powers are more or less diminished, and an inaptitude to any mental exercise is a very common symptom of interstitial hepatitis. These mental symptoms may be very much increased in advanced cases of cirrhosis, in which delirium and convulsions not unfrequently precede a fatal coma. There is a general inability for muscular exercise, and the patient says that he feels lax, and sleep, as a rule, is diminished.

The liver itself is in some cases uncomfortable, and one has the sensation of a heavy weight dragging on the right side, but so long as no active hyperæmia is present it is not painful on pressure or exertion. On careful examination it is as a rule found enlarged, and its lower margin more or less rounded and distinct. In the later stages when contraction comes on its size is diminished. The alimentary system shows certain

changes. Almost invariably there is loss of appetite with more or less discomfort after food. The tongue is as a rule furred, sometimes yellowish, and at other times thickly covered with a white fur. In alcoholic cases morning vomiting and morning diarrhoea are frequent symptoms, but in less marked cases the vomiting may be entirely absent, and the tongue only very slightly furred, but loss of appetite very distinctly marked.

The patients almost invariably complain of flatulency, and the action of the bowels is irregular; as a rule constipation is marked, although now and again it alternates with diarrhoea, the result of the constipation; or when a gastro-intestinal catarrh exists, diarrhoea is a constant symptom. The stools themselves are found to be loaded with urobilin; and if the patient has taken much fat, often the quantity of fat contained in them is increased beyond the normal quantity.

The urine as a rule is dark coloured, the quantity diminished, and often loaded with urates, more especially so when any active hyperæmia is present.

Experiments have shown us that in all probability the liver is the seat of formation—or at any rate the principal seat of formation—of urea, that in the tissues generally carbamide of ammonia is formed, which on reaching the liver is converted into urea, and then excreted by the kidneys.

In cases where the liver tissue is more or less destroyed, as is the case in interstitial hepatitis, one would expect, if such views were correct, the quantity of urea would be diminished, while the quantity of ammonia eliminated in the urine would be increased. This has been found absolutely to be the case.

Hallevorden was the first to describe a case of interstitial hepatitis in which instead of the normal 1 gramme of ammonia per diem, no less than 2·5 grammes was excreted. A healthy individual excretes from 84 to 87 per cent. of the total nitrogen in his urine as urea, while only 2 to 5 per cent. of the total nitrogen is excreted in the urine as ammonia.

In interstitial hepatitis Weintraud found no less than from 4·1 to 11·9 per cent. of the total nitrogen was excreted as ammonia. Other observers have confirmed these results, and it is an aid in the diagnosis of the early stages of interstitial hepatitis

when we find the ammonia increased, and the percentage of nitrogen as urea diminished. In later stages of cirrhosis the urea is markedly diminished, and if the liver tissue is much destroyed the organism has lost the power of forming urea. Those cases where the urea formation is almost abolished are rare, and only occur shortly before death. That this should be the case would naturally be expected when we consider that before the liver could be so destroyed as to stop forming urea, life would be practically impossible.

The only other important condition of the urine in interstitial hepatitis is the almost uniform increase of aromatic sulphates and indican, which are not the result of the liver disease itself, but of the gastro-intestinal changes which accompany, or rather are the cause of the hepatitis.

When the disease has advanced still further, so as to cause contraction of the substance of the liver, the interference with the veins circulating through the liver leads to hæmorrhages either from the stomach, œsophagus, or bowel, and hæmorrhoids are a constant symptom. At the same time ascites supervenes, and diagnosis is in such cases as a rule easy.

Having now briefly considered the symptoms, we will discuss shortly the treatment of this condition, and one of the primary treatments is diet. We should at once forbid all alcohol and highly seasoned foods, so as to avoid as much as possible any tendency to gastro-intestinal catarrh. In all inflammatory processes in any part of the body rest is one of the essentials for cure, and in order to give the liver as much rest as possible, we should limit meat diet to small dimensions, replacing it by fish when necessary, and other light foods. Fat, if found not properly digested in the stools, should be diminished in quantity. Milk is the best food in the early acute stage, and if the symptoms are urgent it is beneficial to limit the diet entirely to milk every three or four hours, until the active inflammation has somewhat subsided, when more varied food may be gradually resumed. Vegetables are useful, as they counteract the tendency to constipation, and their salts seem to be of use in diminishing the inflammatory processes in the liver.

Clothes are most important, so as to keep the blood on the surface of the body as much as possible, and flannels should always be worn, as

any chill will tend to produce an active hyperæmia, and render the symptoms more severe.

As far as possible at first, mental and bodily fatigue should be avoided, but slowly the patient should be encouraged to take exercise, and as the symptoms improve, lead an active, although not overtaxed life. Fresh air and everything which will improve the general tone should be recommended.

The principal symptoms which have to be treated are those of the gastro-intestinal tract. For the nausea which is so often present, bismuth with nitro-hydrochloric acid may be required, and if the nausea is marked, nothing gives such relief as the frequent washing out of the stomach in the early morning with some dilute antiseptic solution.

For the constipation a pill containing nuxvomica and aloes is often of very great service, and to relieve the flatulency some creosote is well added.

In cases where the intestinal putrefaction is very much increased, the washing out of the lower bowels with a copious enema of 2 grains of permanganate of potassium or 2 drachms of boracic acid to the pint is of great service.

The hæmorrhages in the stomach should be treated on general principles. When there is hæmorrhage in the stomach which is with difficulty relieved, I have found in practice it can be often stopped by washing the stomach out with very hot water, and then a solution of perchloride of iron administered by means of a tube. The same holds good for hæmorrhage in the rectum, when other means have failed, by means of enemata.

As far as drugs are concerned, they are of little service, except in treating the bowels and stomach. Use salines to treat the liver while it is acutely inflamed.

In acute hyperæmia of the liver, ammonium chloride or saline purgatives are of great use. When the pain is marked it is often relieved by hot applications or the dry cup, although when marked congestion is present it is as well at once to draw off ten to twenty ounces of blood by means of a small trocar the size of a No. 2 or 3 English catheter.

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Muret and Kaufmann recommended electricity to the abdominal wall, and say that they got marked improvement in ascites by applying the faradic current. This is an easy method to try, but the results in my experience so far have been doubtful.

In all cases where the ascites persists and does not yield readily to drugs, tapping should at once be had recourse to, for when properly carried out it is absolutely free from danger; and although at first tapping is not always successful, when repeated frequently it almost invariably stops the reaccumulation of fluid, as well as which, after the first tapping the drugs which have been found to be useless at the beginning are now often found capable of keeping the ascites down, and provided the liver has not advanced to marked atrophy, the case may entirely recover and the patient live for years, comparatively speaking a healthy man.

Interstitial hepatitis is thus seen to be a larger subject than usually taught, and although I have quoted no cases, so as to make this paper as brief as possible, experience has led me to believe that when properly treated it is perfectly curable, provided it has not advanced to marked atrophy of the liver. In cases of marked atrophy of the liver, with proper treatment we may hope to get some regeneration of liver tissue, in the same way as has been experimentally found in animals by Ponfick, and the patient recovers so far as to lead a healthy life for many years.

ACUTE AND SUBACUTE GONORRHŒA.

BY

CAMPBELL WILLIAMS, F.R.C.S.

GONORRHŒA is a local inflammation due to direct inoculation by a specific germ, the gonococcus. This organism, discovered by Neisser, is sometimes termed the diplococcus, from the fact that it occurs in pairs or in multiples of two. They have a kidney-shaped formation, and measure 1.6μ in length by $.8 \mu$ in breadth. They may be found either free in the pus, or in the bodies of the leucocytes or epithelial cells. The phases of gonorrhœa are classified as acute, subacute, and chronic. In this article reference will only be made to the first two varieties. In the acute form of the disease the amount of constitutional disturbance produced, such as pyrexia, or the occurrence of lymphangitis or lymphadenitis, is, as in other pyogenic conditions, consequent upon the entrance into the system, through the lymph stream, of the products of sepsis, and is proportionate to the virulence of the poison and the quantity absorbed. The general condition of the patient at the time that he contracts the disease has a direct bearing upon the intensity of the subsequent inflammation, as well as the rapidity with which the process spreads along the urethra. The gouty, rheumatic, or strumous diathesis, alcoholism, and the presence of glycosuria or diabetes, are factors which favour the progress and acuteness of an attack. In patients labouring under these conditions there is a greater liability to the acute type of the disease, with early extension of the inflammation backwards, than in those who are not the subjects of these dyscrasiæ. It must be remembered, however, that although gonorrhœa is always present in large cities, there nevertheless seem to be periods when its endemic state reaches to that of an epidemic. That its virulence is not constant in its intensity, the virus seeming at times to be attenuated, and that it occurs in waves of varying severity, has struck me forcibly. As a general rule second attacks savour more of the subacute variety, but speedy implication of the posterior urethra is common with them, and it

when we find the ammonia increased, and the percentage of nitrogen as urea diminished. In later stages of cirrhosis the urea is markedly diminished, and if the liver tissue is much destroyed the organism has lost the power of forming urea. Those cases where the urea formation is almost abolished are rare, and only occur shortly before death. That this should be the case would naturally be expected when we consider that before the liver could be so destroyed as to stop forming urea, life would be practically impossible.

The only other important condition of the urine in interstitial hepatitis is the almost uniform increase of aromatic sulphates and indican, which are not the result of the liver disease itself, but of the gastro-intestinal changes which accompany, or rather are the cause of the hepatitis.

When the disease has advanced still further, so as to cause contraction of the substance of the liver, the interference with the veins circulating through the liver leads to hæmorrhages either from the stomach, œsophagus, or bowel, and hæmorrhoids are a constant symptom. At the same time ascites supervenes, and diagnosis is in such cases as a rule easy.

Having now briefly considered the symptoms, we will discuss shortly the treatment of this condition, and one of the primary treatments is diet. We should at once forbid all alcohol and highly seasoned foods, so as to avoid as much as possible any tendency to gastro-intestinal catarrh. In all inflammatory processes in any part of the body rest is one of the essentials for cure, and in order to give the liver as much rest as possible, we should limit meat diet to small dimensions, replacing it by fish when necessary, and other light foods. Fat, if found not properly digested in the stools, should be diminished in quantity. Milk is the best food in the early acute stage, and if the symptoms are urgent it is beneficial to limit the diet entirely to milk every three or four hours, until the active inflammation has somewhat subsided, when more varied food may be gradually resumed. Vegetables are useful, as they counteract the tendency to constipation, and their salts seem to be of use in diminishing the inflammatory processes in the liver.

Clothes are most important, so as to keep the blood on the surface of the body as much as possible, and flannels should always be worn, as

any chill will tend to produce an active hyperæmia, and render the symptoms more severe.

As far as possible at first, mental and bodily fatigue should be avoided, but slowly the patient should be encouraged to take exercise, and as the symptoms improve, lead an active, although not overtaxed life. Fresh air and everything which will improve the general tone should be recommended.

The principal symptoms which have to be treated are those of the gastro-intestinal tract. For the nausea which is so often present, bismuth with nitro-hydrochloric acid may be required, and if the nausea is marked, nothing gives such relief as the frequent washing out of the stomach in the early morning with some dilute antiseptic solution.

For the constipation a pill containing nuxvomica and aloes is often of very great service, and to relieve the flatulency some creosote is well added.

In cases where the intestinal putrefaction is very much increased, the washing out of the lower bowels with a copious enema of 2 grains of permanganate of potassium or 2 drachms of boracic acid to the pint is of great service.

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Idiocy ; Linear Craniotomy.

On this idiotic child, 2½ years old, I have performed the operation of linear craniotomy. When brought in he had double optic atrophy and was believed to be blind; he apparently heard; he kept his head retracted rigidly, his limbs rigidly flexed; the thumbs always remained in the palms with the fingers closed upon them. There were no clonic spasms. The head was not

strikingly small, but the facies of a low type of idiot was well marked. He lay just as he was placed, cried a great deal without obvious reason, and swallowed with extreme slowness.

The operation is a very simple one, and has for its object to render the skull yielding and to allow the brain to grow if it will. I thought that this idea could be best carried out by setting free the top of the skull. Accordingly on two occasions (a fortnight intervening), and with every precaution against shock, I turned down one or other side of the scalp from near the mid-line and excised a strip of bone half an inch wide, together with its periosteum, from the side of the skull. The two cuts were connected by a cross cut anteriorly and nearly connected posteriorly. The brain was thus covered by a sort of valve of bone easily depressed by finger pressure. With Keene-Hoffman forceps the operations took twenty to twenty-five minutes, and shock was quite moderate. The wounds healed well. I am not in a position to express any opinion on the question whether the brain fails to grow and thus leads to early synostosis of the bones, or whether the premature synostosis is primary and prevents the brain from growing; but the skull was irregularly and greatly thickened in this case, and undoubtedly abnormal elsewhere than at suture-lines. So far, the results of the operation have been very slight. The nurses report that the child cries much less, and that he does not take quite so long in swallowing. The rigid flexion of the limbs has given place to rigid extension, with marked adductor spasm of the thighs. (A month later no improvement had occurred, and the tendency to flexion of the limbs was again marked. The bone flap had again become fixed by ossification across one at least of the grooves cut in the skull.)

NOTES.

The Treatment of Constipation in Infants.

—The 'Journal des Praticiens' of January 9th, 1897, contains a practical article upon this subject. First, it deals with the local accidents which may produce constipation, calling to mind the fact that purgatives should not be given until we

are confident that umbilical or other herniæ do not exist. It may be, too, that prolapse of the rectum or an anal fissure may be a factor to be considered. It is not to be forgotten, also, that fever and cutaneous eruptions sometimes arise in children as the result of constipation. An important factor to be considered in treating these cases is that of heredity and conformation of the intestine. Children of gouty parents frequently suffer from atonic bowels, and in other cases the intestines and abdominal wall seem to be so relaxed as to predispose to this condition. The employment of sterilised milk also favours constipation, and the administration of farinaceous articles too early in life, by provoking dyspeptic troubles, may either result in diarrhoea or constipation. The question of modifying the diet of children, therefore, is of very great importance. If old enough to receive vegetable substances they should be given Graham bread, which leaves a large residue; the ordinary vegetables, such as string-beans and peas, and from time to time mild laxative substances such as manna or cascara, should be given. Frequent exercise in the open air is also a necessity. Very frequently adding a little sugar to the milk, if the child is fed on sterilised milk, will prevent it being so constipating in its effects. Massage of the abdominal area gently applied for a number of minutes morning and night, the skin being rendered oily by the use of vaselin, is also a method which is not to be forgotten. During the massage the fingers should knead the intestines as much as possible. Castor oil and magnesia, while active in moving the bowels, tend to produce constipation to a greater degree after their effects have passed off, although the author of this article believes that calcined magnesia is a useful substance to overcome dyspepsia, and to move the bowel in certain cases. In other instances suppositories and rectal injections produce the best results, particularly suppositories that are made of glycerin. The quantity of liquid which should be used as an injection varies, but ordinarily one or two ounces is sufficient in young children; and if the bowel is not active cold water may be used in place of warm water, and the action of the injection may be increased by the addition of two to three dessertspoonfuls of oil of sweet almonds.—*Therapeutic Gazette*, June 15th, 1897.

TRACHEAL INJECTIONS IN THE TREATMENT OF LARYNGEAL AND PULMONARY INFLAMMATIONS.

By J. A. THOMPSON, M.D.

THE first physician who left us a record of a systematic study of intra-tracheal medication was Dr. Horace Green of New York. Long before the invention of the laryngoscope he had attained remarkable dexterity in passing a sponge probang, saturated with a solution of nitrate of silver, into either bronchus. His reports of his methods and results were so extraordinary that the New York Academy of Medicine appointed an investigating committee to determine the truth or falsity of his statements. The committee agreed that he could and did employ the method described, but a majority condemned its use. With the invention of the laryngoscope there was no immediate revival of the method of direct medication in the treatment of laryngeal and pulmonary inflammations. It is only within the last few years that occasional reference to this subject can be found in medical literature.

There are several reasons for the slow growth of this manner of treatment in professional favour. The principal one is that few physicians are sufficiently expert in the examination and treatment of the upper air-passages to employ it. As a rule they consider all lesions of the nose and throat as local, with but little influence on the health of the patient. This lack of appreciation of the importance of these conditions is largely responsible for the neglect of direct medication. Another reason for the limited use of intra-tracheal injections is the mistaken opinion that they are necessarily painful and irritating. Our ideas of the sensibility of the tracheal and bronchial mucous membrane have been erroneously deduced from that of the larynx. Below the glottis the nerves of sensation are few, and the membrane is not irritated by solutions much stronger than those we use in the larynx. While these reasons have been potent in preventing the more general knowledge and use of this method, there are other and much better reasons for the general use of direct medication in inflammatory diseases of the larynx, trachea, bronchi, and lungs.

By the method of intra-tracheal injection we get

the direct local action of the medicines on the diseased areas. In bronchiectasis no medicine given by the mouth will prevent the decomposition of the secretions in the dilated bronchi. The odour and the absorption of septic material from them cannot be controlled. A few tracheal injections will usually disinfect the cavities, so that the odour disappears and the temperature becomes normal.

It is possible by intra-tracheal medication to produce a rapid and prolonged general effect. Anæsthesia gives daily and hourly evidence of the rapidity with which volatile medicines are absorbed through the lungs, and their effect on the whole organism. From three to five minutes is a sufficient time for an expert to obtain chloroform anæsthesia. You can produce as rapid an effect, and one much more durable, by injecting into the trachea medicines which volatilise slowly at the temperature of the body. Where a dose of menthol had been given in this way, you will find the surface flushed in five minutes; the cold extremities have become warm, and sometimes the patient breaks out in a profuse perspiration. This stimulating action lasts for hours.

Medicines used by tracheal injections are not changed by passing through the digestive organs into unknown compounds. For this reason we can be more certain of their action. No one would think of treating a tubercular laryngitis by internal medication alone. There is just as much reason for applying medicines of known beneficial local action directly to the lungs, as there is for using them in the larynx.

Where medicines are injected directly into the trachea they have no deleterious effect on the organs of digestion. Our valuable expectorants, such as are used in acute and subacute catarrhal diseases, act injuriously on the stomach and intestines. The name of one class, nauseating expectorants, testifies to the universal recognition of this fact. The cure of a bronchitis by direct medication without interfering with nutrition is an advance in therapeutics.

Direct antisepsis can be secured by local medication. It is not possible to obtain this result by remedies given internally. We are prone to forget that in tuberculosis we are dealing with a mixed infection. In the stage of ulceration and breaking down of tissue infiltrated by tubercular matter, there is always a secondary infection by the germs

of suppuration. It is to these, in all probability, that most of the fever, the night sweats, and the other evidences of sepsis are due. We do not attempt to disinfect a leg ulcer by medicating the stomach. It would be just as rational to do so as it is to attempt to disinfect suppurating cavities in the lung by medicine administered *per os*. In cases where tracheal injections can be tolerated, the antiseptic action of the remedies chosen will be very speedily shown, by the subsidence of the cough, by change in the character of the expectoration, and by the decline in the fever.

The administration of medicines by intra-tracheal injection does not interfere with any other line of treatment. Diseases in other organs may be treated or tonics given, while local treatment of the lungs is being used without any incompatibility.

Where medicines are given by the tracheal rather than by the œsophageal route, we can relieve symptoms in hopeless cases without narcotics. We thus avoid their bad effects on nutrition. We also escape their secondary depressing action on the nervous system. A little menthol injected into the trachea will quiet a cough longer and more effectually than will a quarter of a grain of morphia given hypodermically.

Conditions not affected by constitutional medication can be cured by tracheal injections. Gummata in the lung, which resisted all other treatment, have been cured quickly and easily by direct medication of the suppurating cavities.

There are several conditions necessary for success in this method of treatment. The first essential is that the doctor himself be skilful enough in laryngology to do the work rapidly and delicately. The patient must possess a reasonable amount of self-control, and be willing to follow directions implicitly.

It is probable that the medicines used for this treatment should be such as volatilise slowly at the temperature of the body. They should be soluble in the vehicle employed. The solutions used should not be too irritating. The most serviceable solutions are menthol, 2 per cent.; guaiacol, 1 per cent.; creosote, 1 per cent.; camphor, 2 to 3 per cent. Any of these may be combined. In acute diseases the menthol and camphor solutions are most efficient. In tuberculosis menthol and guaiacol give the greatest relief. Guaiacol gives good results in any septic condition in the lungs

or bronchi. The vehicle used should be one of the light petroleum oils or olive oil. Alcohol and water are too irritating, and produce violent coughing. There is ordinarily no spasm and but little cough or distress after an injection of the above solutions. The dose is from one to four drachms. The diseases in which intra-tracheal medication will be of service must be determined by clinical observation.

I wish to present only a few deductions from my experience with this method of treatment during the last five years. The necessary condensation may make my statements seem too dogmatic. All are based on cases actually treated, and are deductions drawn from results thus obtained.

My first notable success was in pulmonary tuberculosis. In August, 1892, I began treating a woman with every symptom of this disease. Her attacks of coughing were so prolonged and violent that she frequently vomited and was unable to sleep at night. An injection of a solution containing menthol 2 per cent., guaiacol 1 per cent. at 4 o'clock in the afternoon would control the cough, so that her dinner would be retained, and she would get a good night's rest. The cough did not return, as a rule, until the following morning. The patient's symptoms entirely disappeared, and treatment was discontinued. A little more than a year after she ceased treatment she became pregnant, and with the digestive disturbances incident to this condition there was a return of the cough, vomiting, and inability to sleep. The treatment by tracheal injection was resumed, again resulting in apparent cure. The pregnancy was uninterrupted, and the patient has had no further occasion for treatment during the last four years.

In other cases of pulmonary tuberculosis, symptoms have been greatly relieved, but I have no other cures where a sufficient interval has elapsed since treatment to make the statistics worth recording.

A few cases that I have seen, where the tubercular disease was making very slow progress, have not been benefited by tracheal injections. Where the diseased process is a chronic, not an acute one, the symptoms have been aggravated by the remedies used.

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The operation is a very simple one, and has for its object to render the skull yielding and to allow the brain to grow if it will. I thought that this idea could be best carried out by setting free the top of the skull. Accordingly on two occasions (a fortnight intervening), and with every precaution against shock, I turned down one or other side of the scalp from near the mid-line and excised a strip of bone half an inch wide, together with its periosteum, from the side of the skull. The two cuts were connected by a cross cut anteriorly and nearly connected posteriorly. The brain was thus covered by a sort of valve of bone easily depressed by finger pressure. With Keene-Hoffman forceps the operations took twenty to twenty-five minutes, and shock was quite moderate. The wounds healed well. I am not in a position to express any opinion on the question whether the brain fails to grow and thus leads to early synostosis of the bones, or whether the premature synostosis is primary and prevents the brain from growing; but the skull was irregularly and greatly thickened in this case, and undoubtedly abnormal elsewhere than at suture-lines. So far, the results of the operation have been very slight. The nurses report that the child cries much less, and that he does not take quite so long in swallowing. The rigid flexion of the limbs has given place to rigid extension, with marked adductor spasm of the thighs. (A month later no improvement had occurred, and the tendency to flexion of the limbs was again marked. The bone flap had again become fixed by ossification across one at least of the grooves cut in the skull.)

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The Treatment of Constipation in Infants.

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are confident that umbilical or other herniæ do not exist. It may be, too, that prolapse of the rectum or an anal fissure may be a factor to be considered. It is not to be forgotten, also, that fever and cutaneous eruptions sometimes arise in children as the result of constipation. An important factor to be considered in treating these cases is that of heredity and conformation of the intestine. Children of gouty parents frequently suffer from atonic bowels, and in other cases the intestines and abdominal wall seem to be so relaxed as to predispose to this condition. The employment of sterilised milk also favours constipation, and the administration of farinaceous articles too early in life, by provoking dyspeptic troubles, may either result in diarrhoea or constipation. The question of modifying the diet of children, therefore, is of very great importance. If old enough to receive vegetable substances they should be given Graham bread, which leaves a large residue; the ordinary vegetables, such as string-beans and peas, and from time to time mild laxative substances such as manna or cascara, should be given. Frequent exercise in the open air is also a necessity. Very frequently adding a little sugar to the milk, if the child is fed on sterilised milk, will prevent it being so constipating in its effects. Massage of the abdominal area gently applied for a number of minutes morning and night, the skin being rendered oily by the use of vaselin, is also a method which is not to be forgotten. During the massage the fingers should knead the intestines as much as possible. Castor oil and magnesia, while active in moving the bowels, tend to produce constipation to a greater degree after their effects have passed off, although the author of this article believes that calcined magnesia is a useful substance to overcome dyspepsia, and to move the bowel in certain cases. In other instances suppositories and rectal injections produce the best results, particularly suppositories that are made of glycerin. The quantity of liquid which should be used as an injection varies, but ordinarily one or two ounces is sufficient in young children; and if the bowel is not active cold water may be used in place of warm water, and the action of the injection may be increased by the addition of two to three dessertspoonfuls of oil of sweet almonds.—*Therapeutic Gazette*, June 15th, 1897.

There was no metrorrhagia or loss during the fortnight's interval. The weakness consequent upon this became so great that at last she had to spend much of her time in bed, and she got excessively anæmic. Two years ago she noticed that she began to get stout about the abdomen, and that she was losing flesh elsewhere. No tumour was discovered, however, until she was admitted into this hospital three months ago, on account of uterine hæmorrhage. Dr. Black kept her in bed, and put her on ergot. This arrested the loss, and she went out. But with the next period the bleeding came on as profusely as ever, and it became obvious that something radical must be done to control it. Moreover, the tumour was growing. The state of matters was that she had a perfectly smooth, pear-shaped swelling, which rose above the pubes in the mid-line to some two or three fingers' breadth above the umbilicus. It was a little higher on the right side than on the left. The tumour seemed fixed, but it was not certain whether the rigidity of the muscles did not account for that. It was so soft that, in some of the notes, it is described as semi-fluctuating. *Per vaginam* you could reach the cervix high up, drawn up by enlargement of the fundus—so much so that the anterior lip had disappeared. Posteriorly there was still a small fornix remaining. The cervix was normal, and there was no growth starting from it. This we were, of course, glad to find, for in cases of hysterectomy in which the broad ligament is opened up by the tumour there is very free bleeding, which you cannot see to stop until you have got the tumour out, and a great deal of blood may be lost before this is effected. We next found that there were no bladder or rectal symptoms; the urine was normal, and there was nothing to indicate that there was pressure on the ureters. The sound passed four to six inches, apparently into the centre of the mass, leading to the diagnosis (faulty, as you have seen) of diffuse fibroid thickening of the uterus.

On March 13th I did an abdominal hysterectomy, and there was nothing unusual about the operation. I had to make a long incision at once from above the umbilicus down to the pubis. The umbilicus was too deep for thorough disinfection, so I removed it entirely. Through this incision I could just squeeze the tumour. The recti remained rigid in spite of the anæsthetist's

efforts, and rendered the operation more difficult by contracting the space I had to work in. As the tumour came out I saw a number of large veins which came off from the right of the uterus in the direction of the ovarian artery. They were tied in two places and cut between, and that let me into the broad ligament. I divided the peritoneum until I had reached and tied the uterine artery on the same side. On the left side the ovary lay high, and was closely bound to the psoas, so that with the rigid muscles I could not tie the ovarian plexus outside it. I applied two ligatures to the veins coming from the uterus just inside the ovary, and divided the broad ligament here. I intended to go back for that ovary, but I did not do so, and it may turn out to have been a good omission. I now got down on the left side of the large tumour and secured the left uterine artery. Lastly, I raised anterior and posterior flaps, chiefly of peritoneum, and removed the uterus by cutting across at about the level of the internal os. Very little blood was lost. All the vessels were tied, and then I sewed up from one ovarian pedicle to the other across the stump of the uterus. The flaps were brought together by deep stitches, and the peritoneum was sewn up again, so that there was a transverse line of stitching right across the pelvis. Everything was restored to its proper place, and the abdominal wall was closed by three layers of stitches. The patient has done most satisfactorily, in spite of the fact that she had a very foul vaginal discharge, which was clearly a source of danger.

Later on two or three slight rises of temperature caused me to make an examination, when I found that the stump of the uterus was fixed, and that a considerable exudation had formed, chiefly posterior to it. Under rest and douches this subsided, and she left the hospital greatly improved in every way.

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TRACHEAL INJECTIONS IN THE TREATMENT OF LARYNGEAL AND PULMONARY INFLAMMATIONS.

By J. A. THOMPSON, M.D.

THE first physician who left us a record of a systematic study of intra-tracheal medication was Dr. Horace Green of New York. Long before the invention of the laryngoscope he had attained remarkable dexterity in passing a sponge probang, saturated with a solution of nitrate of silver, into either bronchus. His reports of his methods and results were so extraordinary that the New York Academy of Medicine appointed an investigating committee to determine the truth or falsity of his statements. The committee agreed that he could and did employ the method described, but a majority condemned its use. With the invention of the laryngoscope there was no immediate revival of the method of direct medication in the treatment of laryngeal and pulmonary inflammations. It is only within the last few years that occasional reference to this subject can be found in medical literature.

There are several reasons for the slow growth of this manner of treatment in professional favour. The principal one is that few physicians are sufficiently expert in the examination and treatment of the upper air-passages to employ it. As a rule they consider all lesions of the nose and throat as local, with but little influence on the health of the patient. This lack of appreciation of the importance of these conditions is largely responsible for the neglect of direct medication. Another reason for the limited use of intra-tracheal injections is the mistaken opinion that they are necessarily painful and irritating. Our ideas of the sensibility of the tracheal and bronchial mucous membrane have been erroneously deduced from that of the larynx. Below the glottis the nerves of sensation are few, and the membrane is not irritated by solutions much stronger than those we use in the larynx. While these reasons have been potent in preventing the more general knowledge and use of this method, there are other and much better reasons for the general use of direct medication in inflammatory diseases of the larynx, trachea, bronchi, and lungs.

By the method of intra-tracheal injection we get

the direct local action of the medicines on the diseased areas. In bronchiectasis no medicine given by the mouth will prevent the decomposition of the secretions in the dilated bronchi. The odour and the absorption of septic material from them cannot be controlled. A few tracheal injections will usually disinfect the cavities, so that the odour disappears and the temperature becomes normal.

It is possible by intra-tracheal medication to produce a rapid and prolonged general effect. Anæsthesia gives daily and hourly evidence of the rapidity with which volatile medicines are absorbed through the lungs, and their effect on the whole organism. From three to five minutes is a sufficient time for an expert to obtain chloroform anæsthesia. You can produce as rapid an effect, and one much more durable, by injecting into the trachea medicines which volatilise slowly at the temperature of the body. Where a dose of menthol had been given in this way, you will find the surface flushed in five minutes; the cold extremities have become warm, and sometimes the patient breaks out in a profuse perspiration. This stimulating action lasts for hours.

Medicines used by tracheal injections are not changed by passing through the digestive organs into unknown compounds. For this reason we can be more certain of their action. No one would think of treating a tubercular laryngitis by internal medication alone. There is just as much reason for applying medicines of known beneficial local action directly to the lungs, as there is for using them in the larynx.

Where medicines are injected directly into the trachea they have no deleterious effect on the organs of digestion. Our valuable expectorants, such as are used in acute and subacute catarrhal diseases, act injuriously on the stomach and intestines. The name of one class, nauseating expectorants, testifies to the universal recognition of this fact. The cure of a bronchitis by direct medication without interfering with nutrition is an advance in therapeutics.

Direct antiseptics can be secured by local medication. It is not possible to obtain this result by remedies given internally. We are prone to forget that in tuberculosis we are dealing with a mixed infection. In the stage of ulceration and breaking down of tissue infiltrated by tubercular matter, there is always a secondary infection by the germs

of suppuration. It is to these, in all probability, that most of the fever, the night sweats, and the other evidences of sepsis are due. We do not attempt to disinfect a leg ulcer by medicating the stomach. It would be just as rational to do so as it is to attempt to disinfect suppurating cavities in the lung by medicine administered *per os*. In cases where tracheal injections can be tolerated, the antiseptic action of the remedies chosen will be very speedily shown, by the subsidence of the cough, by change in the character of the expectoration, and by the decline in the fever.

The administration of medicines by intra-tracheal injection does not interfere with any other line of treatment. Diseases in other organs may be treated or tonics given, while local treatment of the lungs is being used without any incompatibility.

Where medicines are given by the tracheal rather than by the oesophageal route, we can relieve symptoms in hopeless cases without narcotics. We thus avoid their bad effects on nutrition. We also escape their secondary depressing action on the nervous system. A little menthol injected into the trachea will quiet a cough longer and more effectually than will a quarter of a grain of morphia given hypodermically.

Conditions not affected by constitutional medication can be cured by tracheal injections. Gummata in the lung, which resisted all other treatment, have been cured quickly and easily by direct medication of the suppurating cavities.

There are several conditions necessary for success in this method of treatment. The first essential is that the doctor himself be skilful enough in laryngology to do the work rapidly and delicately. The patient must possess a reasonable amount of self-control, and be willing to follow directions implicitly.

It is probable that the medicines used for this treatment should be such as volatilise slowly at the temperature of the body. They should be soluble in the vehicle employed. The solutions used should not be too irritating. The most serviceable solutions are menthol, 2 per cent.; guaiacol, 1 per cent.; creosote, 1 per cent.; camphor, 2 to 3 per cent. Any of these may be combined. In acute diseases the menthol and camphor solutions are most efficient. In tuberculosis menthol and guaiacol give the greatest relief. Guaiacol gives good results in any septic condition in the lungs

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I wish to present only a few deductions from my experience with this method of treatment during the last five years. The necessary condensation may make my statements seem too dogmatic. All are based on cases actually treated, and are deductions drawn from results thus obtained.

My first notable success was in pulmonary tuberculosis. In August, 1892, I began treating a woman with every symptom of this disease. Her attacks of coughing were so prolonged and violent that she frequently vomited and was unable to sleep at night. An injection of a solution containing menthol 2 per cent., guaiacol 1 per cent. at 4 o'clock in the afternoon would control the cough, so that her dinner would be retained, and she would get a good night's rest. The cough did not return, as a rule, until the following morning. The patient's symptoms entirely disappeared, and treatment was discontinued. A little more than a year after she ceased treatment she became pregnant, and with the digestive disturbances incident to this condition there was a return of the cough, vomiting, and inability to sleep. The treatment by tracheal injection was resumed, again resulting in apparent cure. The pregnancy was uninterrupted, and the patient has had no further occasion for treatment during the last four years.

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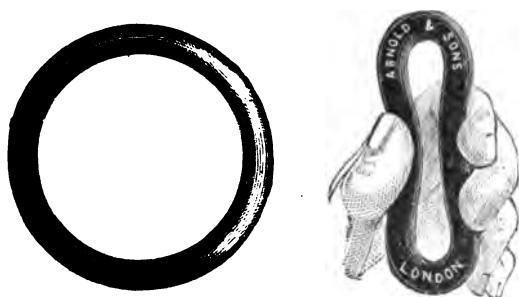
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sufficiently thick, and the spring sufficiently strong to retain its circular shape. It should always be removed at night and replaced in the morning by the patient. The action of the ring differs from the Hodge in that it gets no support from the vagina, but finds its resting point on the walls of the pelvis, the spine and tuberosities of the ischium,



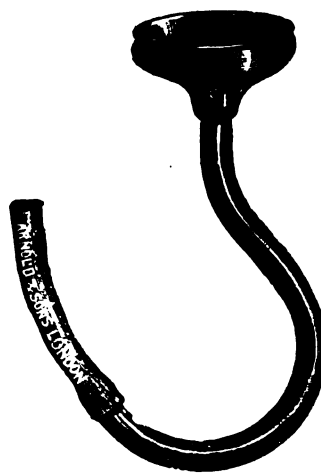
and the sacro-sciatic ligaments extending backwards from them. Its action is that of the Hodge above described. By drawing the vagina backward it draws the cervix in the same direction, and so tilts the fundus forwards. It does not materially raise the uterus, but it prevents further descent, and is a very useful pessary in practice. I often use it in preference to a Hodge when it fits the vagina comfortably. It is quite unsuitable where the vagina retains its shape, not having been distended by childbirth or some other cause. In cases of complete prolapse of the uterus, ring pessaries are seldom sufficient. The invagination of the vagina and the pressure from above almost invariably force out the ring and uterus together. The ordinary sizes of the ring pessary used are $2\frac{1}{2}$, $2\frac{3}{4}$, and 3 inches in diameter. A ring above this size frequently causes inconvenience by pressing on the rectum.

In complete prolapse Zwanck's pessary will be found more efficacious. There are endless varieties of this pessary, but the one I have here is certainly the best. It takes its rest upon the same part of the bony pelvis as the ring pessary, and like it is only suitable when the vagina has been dilated and lost its original shape and tone. No Zwanck's pessary, however small, can be inserted into a nulliparous vagina. It should always be removed at night. The patient after a little practice will have no difficulty in managing it for herself. It should be freely moveable in the pelvis, not press-

ing on the bony walls, or causing tension of the vagina. The sizes most frequently required are

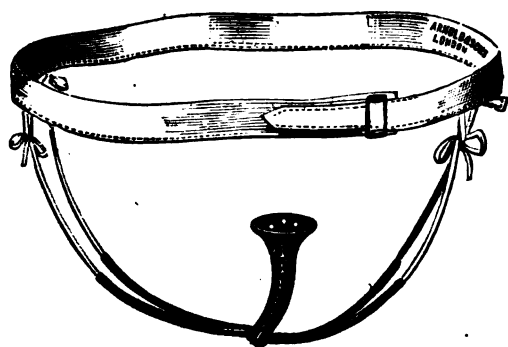


3, $3\frac{1}{4}$, and $3\frac{1}{2}$ inches. In the event of the pelvic walls affording no resting-place for the wings of the pessary, we must have recourse to the cup and stem pessary, or some modification of Cutter's pessary which obtains its support from the waist-belt. No little trouble is often expe-



rienced in supporting the prolapse of elderly women when the vagina has become atrophied, and will not allow the introduction of a pessary sufficiently large to rest upon the pelvic wall. I do not think I need take up your time with a discussion on prolapse. For all practical pur-

poses it is sufficient to know that retroflexion and retroversion are the early stages of descent of the uterus, and that if it continues to progress the appearance of the uterus outside the vulva is only a matter of time. In descent of the pelvic floor, the whole or a part only may be affected. In



some cases the anterior vaginal wall and bladder are the first to appear outside the vulva (cystocele), then the cervix, and finally the posterior vaginal wall. My own experience, however, is that the cervix is generally the first part to appear at the vulvar outlet. The posterior wall in its lower part (last inch) is closely adherent to the perineal body, and rarely completely descends.

You will not be likely to mistake hypertrophic elongation of the cervix for prolapse of the uterus.

I have endeavoured to give you as briefly as possible my own views on the use of pessaries. I should add I do not make the same extensive use of pessaries for minor degrees of displacement that I formerly did. Pessaries are undoubtedly very objectionable instruments for many reasons; and when we consider how very little suffering is sometimes caused by prolapse in the major degree, a suspicion arises that the importance attached to the minor forms is somewhat overestimated. Where the indications are clear the best results attend their use, but it does not necessarily follow because a uterus is retroflexed that the symptoms complained of are due to that fact. Neither is it possible with any pessary devised to restore every case of flexion and maintain it in position. The less we talk to our patients about "misplacement" the better. It is a still greater mistake to insert a pessary unnecessarily. The female mind becomes so impressed with the idea of the necessity for the artificial support,

whether it does any good or not, that the subsequent conduct of the case becomes a matter of real difficulty.

I must add I remove almost as many pessaries as I insert; and on a first visit make a point of removing a pessary to watch the effects on the next occasion. I find many have been inserted without the smallest pretence of reason.

A lecture of this kind would probably be considered incomplete without a brief reference to the operative measures available in these cases. We are passing through an era of gynaecology which may appropriately be termed an "operative era." There is a danger that the interest excited by the brilliant results of modern abdominal surgery may cause us to lose sight of and underrate the value of some of our former means of treatment. But there is ample scope for both, the more recent operative work and what there is of value in our old methods of treatment. I would point out, moreover, that very many recognised gynaecological operations have fallen into disuse, and very many of the more recent operations are still waiting to have their value proved.

Shortening the round ligaments (Alexander's operation) is the one which perhaps has aroused most attention. The round ligaments are exposed on either side by an incision upwards and outwards from the pubic spine, in the direction of the inguinal canal. The incision is about two inches in length, depending upon the amount of fat which covers the parietes. When found the ligaments are separated from their connections in the inguinal canal and gently drawn forwards. They are then secured to the pillars of the external abdominal ring by two or three fine buried sutures. Excellent results are claimed by Alexander and one or two others for this operation. Its disadvantages are that the ligaments are often very difficult to find, and this difficulty necessitates a longer incision. The tendency to hernia is then increased. It is a necessary condition, moreover, that the uterus should be moveable. This latter appears to be its most serious defect. It is just in those cases where the uterus is free from adhesions and freely moveable that a Hodge pessary answers all requirements. There seems, then, to be really only a limited scope for the operation, viz. in those cases where for some reason, there being no complications, a Hodge pessary fails to act.

In prolapse I cannot see that shortening the round ligaments can be of any use. The normal structures having given way, either from debility or the strain imposed upon them, the whole pelvic floor descends, and it is impossible to conceive that all this amount of mischief can be rectified by two unimportant ligaments, attached by two or three fine sutures to the external abdominal ring.

Dr. Abbe (New York) says that when the ligament has been drawn out and cut off at the external abdominal ring, it is hopeless to expect it permanently to seal itself in the elastic tissue of the new inguinal scar with the uterus dragging constantly upon it. He therefore utilises the round ligament itself for the double purpose of sewing up the inguinal canal, and securing the ligament immovably.

"Primary union invariably occurs after the operation, and in thin subjects the buried natural suture can be felt for weeks. The results are all that can be desired." I have no personal experience of this plan, but feel somewhat sceptical that a self-respecting ligament would allow itself to be treated in such a way.

Hysteropexy, or Ventral Fixation.—This operation is less scientific than the preceding, but it possibly has some advantages. The peritoneum may be opened or otherwise, the condition of the uterus and appendages ascertained if necessary, and the fundus uteri attached by means of sutures to the linea alba. It appears to me that, like Alexander's operation, it might have a limited scope in cases of retroflexion which cannot be otherwise treated, but that it must be useless in prolapse.

The adhesions so formed would probably soon stretch, and buried sutures are never to be depended upon.

Vaginal Fixation.—In this proceeding the peritoneal pouch between the uterus and bladder is opened up, as in the anterior incision for vaginal hysterectomy. The retroverted uterus is anteverted into the wound, fixed there, and the vaginal flaps closed over it. This is the so-called vaginal fixation of the uterus.

Apart from the immediate result of these operations it is important, when they are performed during the child-bearing period, to ascertain what consequences would be should pregnancy

occur. According to Dr. Noble (Philadelphia) the results are simply disastrous. In one fourth of the cases in which pregnancy occurred abortion was the result, and in those which went to full time serious obstetrical complications arose, necessitating delivery by forceps, version, craniotomy, and even Cæsarean section. He regarded it, therefore, as unjustifiable during the age of child-bearing.

Vaginal extirpation of the prolapsed uterus is advised by some, and has been carried out frequently in America and on the Continent. It is not practised in this country. I should doubt if it cured the prolapse altogether, and I think would further weaken the pelvic floor.

Porro's Operation.—A case occurred to me quite recently in which an elderly woman, with both ovaries cystic and distending the abdomen, for which the operation was undertaken, suffered also from prolapse of considerable size. Instead of ligating the pedicles and returning them, I drew up the uterus, which was atrophied, and included the whole in the wire loop, fixing it outside the parietes. I shall follow the case with interest to see the ultimate result. She will have six weeks in the recumbent position, in which the prolapsed organs may regain their tone, if they will, and there will be no question as to the union of the stump with the abdominal parietes.

I have another case in hand, in which the patient is only 42 years of age, the uterus enlarged and filling the pelvis. A Zwanck's pessary supports the prolapse. I believe removal of the uterus will afford this patient much relief. In such a case Porro's operation appears to me to have decided advantages over vaginal extirpation, and in suitable cases to be a decidedly good proceeding.

Hepatic Complications of Typhoid Fever.

—Dr. Osler considers this a rare complication, and when it occurs is apt to be mistaken for appendicitis. He reported a case which had been operated upon for appendicitis, and on opening the abdomen the appendix was found to be normal. The gall-bladder was much enlarged and inflamed, and contained a quantity of fluid, which was withdrawn, and the colon bacillus found to be present. An operation for the removal of a gall-stone was performed some time afterwards.

Pediatrics, June 1st, 1897.

DEMONSTRATION OF CASES AT CHARING CROSS HOSPITAL,

By STANLEY BOYD, M.B.Lond., F.R.C.S.,
Surgeon to the Hospital.

Specimen of Cancer of Breast (from the case reported in CLINICAL JOURNAL, June 2nd).

GENTLEMEN,—You will remember that when we last went round the wards I showed you an extremely bad and rapid case of cancer of the breast, with great swelling of the arm and side of the trunk. I then considered the advisability of employing the recently suggested treatment of inoperable mammary cancer by double oöphorectomy; but I finally decided that the disease was in this case too far advanced to allow any reasonable hope of success. At that time the patient was suffering from dyspnœa, due, as I believed, to fluid in the left pleura. To relieve her, we drew off thirty ounces of fluid, which contained no trace of blood. The fluid returned rapidly, and we endeavoured to tap again; but although I put the whole length of an aspirator needle into the chest wall, I could not reach the fluid, so thick was the wall from œdema. The patient gradually sank from exhaustion and dyspnœa, and died in two to three weeks. I now have the breast to show you. You will remember that the skin was becoming vascular and infiltrated with extreme rapidity. There was hardly any discoloration on admission, but later we noticed it spreading every day, from the left breast across the mid-line. You can now see the cancerous infiltration of the skin with the naked eye; it is from a third to half an inch thick. There was one mass of cancer running thence right through the breast, infiltrating both pectorals, and adhering directly to the chest wall. Here the solid mass of cancer broke up, and there were discrete nodules in the intercostal muscles. On opening the chest and looking into the pleura, which was full of fluid, there were many nodules on the external parietal pleural surface. The apex of the left lung was adherent to the pleura, and on the lung side of this adhesion there was a small mass of cancer as large as the end of a thumb. There were no other secondary visceral growths found. The left lung was absolutely collapsed,

and weighed $9\frac{1}{2}$ oz. The right weighed 24 oz., showing what a lot of blood and fluid is contained in the ordinary tissues. Next as to the cause of the swelling of the arm—of the so-called lymphœdema, a name which might seem to imply that it was due to obstruction in the lymph current rather than in the veins. But we all know that obstruction sufficient to cause œdema is much more common in veins than in lymphatics. If the lymphatic glands on one side of the neck be completely removed, as far as we can, œdema does not occur; indeed, a very free removal may be practised upon both sides without ill result. The only instance I can remember in which I have been led to suppose that the removal of lymphatic glands led to œdema was a case of very early cancer, in which I removed the breast very freely, together with all the axillary glands I could find—none being obviously diseased. That woman developed œdema of the arm, and although I was unable to prove it by excluding venous thrombosis, it did seem likely that this was a case in which removal of a large number of acting glands resulted in œdema. When we remove a lot of tubercular glands, the reason why untoward consequences do not, as a rule, arise is that their removal has really been gradual and due to the disease, so that when they are taken from the body, they are not active glands, and a collateral lymph circulation has presumably been established. But, in the case I have mentioned above, I removed apparently healthy glands suddenly, and a long persistent œdema of the arm resulted. In the case from which the specimen which I am showing you was taken, I dissected out all the vessels up to the collar bone. The axillary artery high up was very considerably constricted in the cancer tissue. I slit up the veins, and at three points between the lower part of the axilla and the junction of the subclavian and jugular veins there were rounded cancer masses growing into the axillary vein, greatly narrowing the channel, but still covered with epithelium. Three obstructions of this character would obviously oppose a great difficulty to the return of blood, and would alone, as I think form a sufficient cause of the lymphœdema; though, doubtless, innumerable lymphatics were blocked besides. Probably the swelling would have been still more extreme if the artery also had not been constricted.

Specimen of Fibro-myoma Uteri.

I also wish to show you here a tumour removed from a case which we will look at upstairs. You see it is a uterus as large as if six months pregnant; it contains a large, soft, sessile fibroid, which grew from the fundus and anterior wall into its cavity. The woman was blanched by repeated hæmorrhage; no medical treatment had long benefited her, and hysterectomy was indicated, which I performed. The specimen is much smaller than when I removed it. In the first place enough fluid came from it to fill a soup plate, and it has been left in formalin ever since, which constricts the tissues.

Ossification of Tendon of Adductor Longus.

I want you to just look at this woman, æt. 59. She was knocked down by a hansom cab on December 14th. She does not know how she fell, but the principal injury was to the right ankle, and she was bruised in various parts. Two weeks after she was in hospital she discovered that she had a swelling about the pubes, and she now says that it hurts her when she walks, and when she moves the muscles of the leg. On examination you will find that there is a pointed, bony swelling one and a half inches long, which starts from the pubic bone, just on the right of the mid-line. The tendon of the adductor longus stands out clearly when I make her contract the muscle, and the swelling lies in the tendon of origin of the adductor. It is apparently an ossification in this tendon, although on the other side there is no indication of anything of the kind, and tendons usually ossify symmetrically. What is the reason she is suffering pain from it now? We are all familiar with the cases in which, when persons become aware that they have got something the matter with them, they find also that it becomes painful. Sir James Paget tells of a lady who had a cancer of the breast, which she had discovered a few days before she went to see him. In reply to his question, "Is it painful?" she replied, "Only since I noticed it." The only other solution of this present case would be that the woman had sustained a fracture of the pelvis of a serious nature, with displacement of fragments, and that it was not discovered when she was in hospital and had no need to walk about. But I'll be able to feel that the upper rim of the

pelvis shows no trace of deformity or of callus, and the pubic arch immediately below the bony spine appears to be normal. So I think we may safely regard it as a case of ossification in the adductor longus tendon on one side only.

A Submaxillary Nævus with misleading History.

This woman, æt. 38, has a swelling below her jaw. The history is that for twenty-four years she has noticed a swelling in the left submaxillary region. When she came in we thought it was about the size of a large walnut. She says that with tolerable regularity it increases in size for two or three days, and then diminishes, the diminution being accompanied by a sort of bursting and discharge into the mouth, which she compares to saliva. The discharge is so free that she has to use four or five handkerchiefs to spit into. About once in every two or three months the discharge is yellowish and tastes bad, but ordinarily it is just like saliva. This discharge, she thinks, enters the mouth far back under the tongue. When the swelling reaches its fullest point there is some little pain, then the discharge takes place and everything quiets down. We found, on her admission, that the swelling was very soft, slightly lobulated on the surface, and that it could be pushed about somewhat beneath the skin, when its lobulation became visible. The skin was normal, except that at two spots it had a thin, bluish, translucent look. We did not notice any swelling up after pressure like a nævus. She was not long under observation, but one observer at least thought he had noted diminution in size. The whole history was so complete that the diagnosis of a cyst in connection with the submaxillary gland seemed an obvious one. We could not find any abnormal opening into the mouth, and supposed that the discharge occurred through Wharton's duct. I proposed to cut down from the outside upon the cyst, dissect it out, endeavour to find the opening in the mouth and close it. My surprise was very considerable when, at my first incision, I found that I had to deal with an ordinary cavernous nævus, occupying the whole submaxillary region superficial to the salivary gland, which was quite normal. By the sacrifice of a little blood I got all the way round it and removed it.

Nævous tissue will always bleed if you cut *into* it; oozing is then continuous and rapid. Therefore, in removing a nævus always keep *outside* in the healthy tissues around it. I show you the nævus which was taken away that you may note its sponge-like, trabecular structure, and be able to recognise it in the living subject.

The patient has had no discharge from the mouth since the operation. I do not know what "suggested" this discharge to her, but it would seem to have had a subjective cause. This case shows how much we may sometimes be misled by a history, and tends to breed scepticism with regard to patients' statements; yet every now and then, on the other hand, we have to regret not having paid more attention to a particular history.

Tubercular Elbow; Excision.

This little girl, æt. 14, had a history of pain and swelling about the elbow for ten months before coming in here last February. She was treated by incomplete rest and iodine painting. Just before Christmas, 1896, an abscess burst on the inner side of the elbow above the epicondyle, and since then another has burst. After Christmas a swelling formed in the axilla, and increased rapidly. When she was admitted we found her elbow diffusely and greatly swollen, the swelling extending from three inches above the joint to two inches below it. There were two sinuses just above the inner condyle, opening in the line of the scar left by incision of the abscess which first formed. On the outer side between the olecranon and the condyle there was a red fluctuating area, and the skin was tense and shiny. Pressure here caused oozing of pus from the internal sinuses. The arm was fixed at an angle of about 55°, and pronation and supination were reduced to 5°. The axillary glands formed a subpectoral swelling, which was very obvious to the eye. Altogether, therefore, there was a very unpromising state of matters. I immediately opened the abscess on the outer side of the olecranon on February 8th, and on February 13th I did the regular operation of excision of the elbow. First of all I opened up the two sinuses on the inner side. On reaching the humerus I found that there was a large hole leading into it, and that its lower end was much "expanded." I cleaned out the cavity, and then found that there was a way down into the elbow-joint. It was

clearly by this track that the pus which escaped on pressure over the head of the radius had passed through the joint. I opened the joint by the ordinary long median posterior incision, separated the triceps from the olecranon, and then cut off the top of the ulna. I found that the whole of the olecranon process, as I have shown in this drawing, was tubercular, and I had to gouge out the shaft below the coronoid process to make reasonably sure that all disease had been removed. The radius was only superficially affected, and I removed its head with a knife. Lastly, I cut away the whole lower joint surface of the humerus, and through that large hole I looked into the cavity of the expanded shaft and cleared tubercular tissue out of quite the lower fourth of the bone. Nothing but a mere shell was left here, and the part as a whole looked almost too bad to be saved. I treated by plugging the cavity firmly with iodoform gauze,—a somewhat painful, but most useful form of dressing. There is now practically no discharge, and the large wound is now healing up in such an astonishing way that to avoid a stiff elbow we have to employ daily forcible movements. We are further trying to develop the wasted muscles by regular faradism.

The primary source of disease having been removed, the large mass of glands in the axilla has all but gone—very much to my surprise, I must confess. I have never seen such a mass of tubercular glands disappear; and if I had not had a long operation on the elbow I should certainly have removed the axillary glands at the same time.

The explanation probably is that the resistance of the gland tissues was of no avail against the tubercle bacilli so long as fresh supplies of organisms (both tubercular and septic) were constantly being conveyed to them by the lymph-stream; but when this was prevented by the treatment adopted, the tissues were able to struggle successfully against the organisms present in them.

Fibro-myoma Uteri; Hysterectomy.

The next patient I want to show you is a single woman, æt. 34, a servant, whom I mentioned before we came into the ward in connection with the specimen of uterine fibroid which I showed you. She came complaining of losing too much at her periods, the discharge lasting latterly for a fortnight at a time, and being very profuse.

There was no metrorrhagia or loss during the fortnight's interval. The weakness consequent upon this became so great that at last she had to spend much of her time in bed, and she got excessively anæmic. Two years ago she noticed that she began to get stout about the abdomen, and that she was losing flesh elsewhere. No tumour was discovered, however, until she was admitted into this hospital three months ago, on account of uterine hæmorrhage. Dr. Black kept her in bed, and put her on ergot. This arrested the loss, and she went out. But with the next period the bleeding came on as profusely as ever, and it became obvious that something radical must be done to control it. Moreover, the tumour was growing. The state of matters was that she had a perfectly smooth, pear-shaped swelling, which rose above the pubes in the mid-line to some two or three fingers' breadth above the umbilicus. It was a little higher on the right side than on the left. The tumour seemed fixed, but it was not certain whether the rigidity of the muscles did not account for that. It was so soft that, in some of the notes, it is described as semi-fluctuating. *Per vaginam* you could reach the cervix high up, drawn up by enlargement of the fundus—so much so that the anterior lip had disappeared. Posteriorly there was still a small fornix remaining. The cervix was normal, and there was no growth starting from it. This we were, of course, glad to find, for in cases of hysterectomy in which the broad ligament is opened up by the tumour there is very free bleeding, which you cannot see to stop until you have got the tumour out, and a great deal of blood may be lost before this is effected. We next found that there were no bladder or rectal symptoms; the urine was normal, and there was nothing to indicate that there was pressure on the ureters. The sound passed four to six inches, apparently into the centre of the mass, leading to the diagnosis (faulty, as you have seen) of diffuse fibroid thickening of the uterus.

On March 13th I did an abdominal hysterectomy, and there was nothing unusual about the operation. I had to make a long incision at once from above the umbilicus down to the pubis. The umbilicus was too deep for thorough disinfection, so I removed it entirely. Through this incision I could just squeeze the tumour. The recti remained rigid in spite of the anæsthetist's

efforts, and rendered the operation more difficult by contracting the space I had to work in. As the tumour came out I saw a number of large veins which came off from the right of the uterus in the direction of the ovarian artery. They were tied in two places and cut between, and that let me into the broad ligament. I divided the peritoneum until I had reached and tied the uterine artery on the same side. On the left side the ovary lay high, and was closely bound to the psoas, so that with the rigid muscles I could not tie the ovarian plexus outside it. I applied two ligatures to the veins coming from the uterus just inside the ovary, and divided the broad ligament here. I intended to go back for that ovary, but I did not do so, and it may turn out to have been a good omission. I now got down on the left side of the large tumour and secured the left uterine artery. Lastly, I raised anterior and posterior flaps, chiefly of peritoneum, and removed the uterus by cutting across at about the level of the internal os. Very little blood was lost. All the vessels were tied, and then I sewed up from one ovarian pedicle to the other across the stump of the uterus. The flaps were brought together by deep stitches, and the peritoneum was sewn up again, so that there was a transverse line of stitching right across the pelvis. Everything was restored to its proper place, and the abdominal wall was closed by three layers of stitches. The patient has done most satisfactorily, in spite of the fact that she had a very foul vaginal discharge, which was clearly a source of danger.

Later on two or three slight rises of temperature caused me to make an examination, when I found that the stump of the uterus was fixed, and that a considerable exudation had formed, chiefly posterior to it. Under rest and douches this subsided, and she left the hospital greatly improved in every way.

Idiocy ; Linear Craniotomy.

On this idiotic child, 2½ years old, I have performed the operation of linear craniotomy. When brought in he had double optic atrophy and was believed to be blind; he apparently heard; he kept his head retracted rigidly, his limbs rigidly flexed; the thumbs always remained in the palms with the fingers closed upon them. There were no clonic spasms. The head was not

strikingly small, but the facies of a low type of idiot was well marked. He lay just as he was placed, cried a great deal without obvious reason, and swallowed with extreme slowness.

The operation is a very simple one, and has for its object to render the skull yielding and to allow the brain to grow if it will. I thought that this idea could be best carried out by setting free the top of the skull. Accordingly on two occasions (a fortnight intervening), and with every precaution against shock, I turned down one or other side of the scalp from near the mid-line and excised a strip of bone half an inch wide, together with its periosteum, from the side of the skull. The two cuts were connected by a cross cut anteriorly and nearly connected posteriorly. The brain was thus covered by a sort of valve of bone easily depressed by finger pressure. With Keene-Hoffman forceps the operations took twenty to twenty-five minutes, and shock was quite moderate. The wounds healed well. I am not in a position to express any opinion on the question whether the brain fails to grow and thus leads to early synostosis of the bones, or whether the premature synostosis is primary and prevents the brain from growing; but the skull was irregularly and greatly thickened in this case, and undoubtedly abnormal elsewhere than at suture-lines. So far, the results of the operation have been very slight. The nurses report that the child cries much less, and that he does not take quite so long in swallowing. The rigid flexion of the limbs has given place to rigid extension, with marked adductor spasm of the thighs. (A month later no improvement had occurred, and the tendency to flexion of the limbs was again marked. The bone flap had again become fixed by ossification across one at least of the grooves cut in the skull.)

NOTES.

The Treatment of Constipation in Infants.

—The 'Journal des Praticiens' of January 9th, 1897, contains a practical article upon this subject. First, it deals with the local accidents which may produce constipation, calling to mind the fact that purgatives should not be given until we

are confident that umbilical or other herniæ do not exist. It may be, too, that prolapse of the rectum or an anal fissure may be a factor to be considered. It is not to be forgotten, also, that fever and cutaneous eruptions sometimes arise in children as the result of constipation. An important factor to be considered in treating these cases is that of heredity and conformation of the intestine. Children of gouty parents frequently suffer from atonic bowels, and in other cases the intestines and abdominal wall seem to be so relaxed as to predispose to this condition. The employment of sterilised milk also favours constipation, and the administration of farinaceous articles too early in life, by provoking dyspeptic troubles, may either result in diarrhoea or constipation. The question of modifying the diet of children, therefore, is of very great importance. If old enough to receive vegetable substances they should be given Graham bread, which leaves a large residue; the ordinary vegetables, such as string-beans and peas, and from time to time mild laxative substances such as manna or cascara, should be given. Frequent exercise in the open air is also a necessity. Very frequently adding a little sugar to the milk, if the child is fed on sterilised milk, will prevent it being so constipating in its effects. Massage of the abdominal area gently applied for a number of minutes morning and night, the skin being rendered oily by the use of vaselin, is also a method which is not to be forgotten. During the massage the fingers should knead the intestines as much as possible. Castor oil and magnesia, while active in moving the bowels, tend to produce constipation to a greater degree after their effects have passed off, although the author of this article believes that calcined magnesia is a useful substance to overcome dyspepsia, and to move the bowel in certain cases. In other instances suppositories and rectal injections produce the best results, particularly suppositories that are made of glycerin. The quantity of liquid which should be used as an injection varies, but ordinarily one or two ounces is sufficient in young children; and if the bowel is not active cold water may be used in place of warm water, and the action of the injection may be increased by the addition of two to three dessertspoonfuls of oil of sweet almonds.—*Therapeutic Gazette*, June 15th, 1897.

TRACHEAL INJECTIONS IN THE TREATMENT OF LARYNGEAL AND PULMONARY INFLAMMATIONS.

By J. A. THOMPSON, M.D.

THE first physician who left us a record of a systematic study of intra-tracheal medication was Dr. Horace Green of New York. Long before the invention of the laryngoscope he had attained remarkable dexterity in passing a sponge probang, saturated with a solution of nitrate of silver, into either bronchus. His reports of his methods and results were so extraordinary that the New York Academy of Medicine appointed an investigating committee to determine the truth or falsity of his statements. The committee agreed that he could and did employ the method described, but a majority condemned its use. With the invention of the laryngoscope there was no immediate revival of the method of direct medication in the treatment of laryngeal and pulmonary inflammations. It is only within the last few years that occasional reference to this subject can be found in medical literature.

There are several reasons for the slow growth of this manner of treatment in professional favour. The principal one is that few physicians are sufficiently expert in the examination and treatment of the upper air-passages to employ it. As a rule they consider all lesions of the nose and throat as local, with but little influence on the health of the patient. This lack of appreciation of the importance of these conditions is largely responsible for the neglect of direct medication. Another reason for the limited use of intra-tracheal injections is the mistaken opinion that they are necessarily painful and irritating. Our ideas of the sensibility of the tracheal and bronchial mucous membrane have been erroneously deduced from that of the larynx. Below the glottis the nerves of sensation are few, and the membrane is not irritated by solutions much stronger than those we use in the larynx. While these reasons have been potent in preventing the more general knowledge and use of this method, there are other and much better reasons for the general use of direct medication in inflammatory diseases of the larynx, trachea, bronchi, and lungs.

By the method of intra-tracheal injection we get

the direct local action of the medicines on the diseased areas. In bronchiectasis no medicine given by the mouth will prevent the decomposition of the secretions in the dilated bronchi. The odour and the absorption of septic material from them cannot be controlled. A few tracheal injections will usually disinfect the cavities, so that the odour disappears and the temperature becomes normal.

It is possible by intra-tracheal medication to produce a rapid and prolonged general effect. Anæsthesia gives daily and hourly evidence of the rapidity with which volatile medicines are absorbed through the lungs, and their effect on the whole organism. From three to five minutes is a sufficient time for an expert to obtain chloroform anæsthesia. You can produce as rapid an effect, and one much more durable, by injecting into the trachea medicines which volatilise slowly at the temperature of the body. Where a dose of menthol had been given in this way, you will find the surface flushed in five minutes; the cold extremities have become warm, and sometimes the patient breaks out in a profuse perspiration. This stimulating action lasts for hours.

Medicines used by tracheal injections are not changed by passing through the digestive organs into unknown compounds. For this reason we can be more certain of their action. No one would think of treating a tubercular laryngitis by internal medication alone. There is just as much reason for applying medicines of known beneficial local action directly to the lungs, as there is for using them in the larynx.

Where medicines are injected directly into the trachea they have no deleterious effect on the organs of digestion. Our valuable expectorants, such as are used in acute and subacute catarrhal diseases, act injuriously on the stomach and intestines. The name of one class, nauseating expectorants, testifies to the universal recognition of this fact. The cure of a bronchitis by direct medication without interfering with nutrition is an advance in therapeutics.

Direct antiseptics can be secured by local medication. It is not possible to obtain this result by remedies given internally. We are prone to forget that in tuberculosis we are dealing with a mixed infection. In the stage of ulceration and breaking down of tissue infiltrated by tubercular matter, there is always a secondary infection by the germs

of suppuration. It is to these, in all probability, that most of the fever, the night sweats, and the other evidences of sepsis are due. We do not attempt to disinfect a leg ulcer by medicating the stomach. It would be just as rational to do so as it is to attempt to disinfect suppurating cavities in the lung by medicine administered *per os*. In cases where tracheal injections can be tolerated, the antiseptic action of the remedies chosen will be very speedily shown, by the subsidence of the cough, by change in the character of the expectoration, and by the decline in the fever.

The administration of medicines by intra-tracheal injection does not interfere with any other line of treatment. Diseases in other organs may be treated or tonics given, while local treatment of the lungs is being used without any incompatibility.

Where medicines are given by the tracheal rather than by the œsophageal route, we can relieve symptoms in hopeless cases without narcotics. We thus avoid their bad effects on nutrition. We also escape their secondary depressing action on the nervous system. A little menthol injected into the trachea will quiet a cough longer and more effectually than will a quarter of a grain of morphia given hypodermically.

Conditions not affected by constitutional medication can be cured by tracheal injections. Gummata in the lung, which resisted all other treatment, have been cured quickly and easily by direct medication of the suppurating cavities.

There are several conditions necessary for success in this method of treatment. The first essential is that the doctor himself be skilful enough in laryngology to do the work rapidly and delicately. The patient must possess a reasonable amount of self-control, and be willing to follow directions implicitly.

It is probable that the medicines used for this treatment should be such as volatilise slowly at the temperature of the body. They should be soluble in the vehicle employed. The solutions used should not be too irritating. The most serviceable solutions are menthol, 2 per cent.; guaiacol, 1 per cent.; creosote, 1 per cent.; camphor, 2 to 3 per cent. Any of these may be combined. In acute diseases the menthol and camphor solutions are most efficient. In tuberculosis menthol and guaiacol give the greatest relief. Guaiacol gives good results in any septic condition in the lungs

or bronchi. The vehicle used should be one of the light petroleum oils or olive oil. Alcohol and water are too irritating, and produce violent coughing. There is ordinarily no spasm and but little cough or distress after an injection of the above solutions. The dose is from one to four drachms. The diseases in which intra-tracheal medication will be of service must be determined by clinical observation.

I wish to present only a few deductions from my experience with this method of treatment during the last five years. The necessary condensation may make my statements seem too dogmatic. All are based on cases actually treated, and are deductions drawn from results thus obtained.

My first notable success was in pulmonary tuberculosis. In August, 1892, I began treating a woman with every symptom of this disease. Her attacks of coughing were so prolonged and violent that she frequently vomited and was unable to sleep at night. An injection of a solution containing menthol 2 per cent., guaiacol 1 per cent. at 4 o'clock in the afternoon would control the cough, so that her dinner would be retained, and she would get a good night's rest. The cough did not return, as a rule, until the following morning. The patient's symptoms entirely disappeared, and treatment was discontinued. A little more than a year after she ceased treatment she became pregnant, and with the digestive disturbances incident to this condition there was a return of the cough, vomiting, and inability to sleep. The treatment by tracheal injection was resumed, again resulting in apparent cure. The pregnancy was uninterrupted, and the patient has had no further occasion for treatment during the last four years.

In other cases of pulmonary tuberculosis, symptoms have been greatly relieved, but I have no other cures where a sufficient interval has elapsed since treatment to make the statistics worth recording.

A few cases that I have seen, where the tubercular disease was making very slow progress, have not been benefited by tracheal injections. Where the diseased process is a chronic, not an acute one, the symptoms have been aggravated by the remedies used.

My next notable success in this treatment was in pulmonary syphilis. Fortunately this is a rare

trophic rhinitis. The only constant symptom of this condition is nasal obstruction of greater or less degree. Such symptoms as paroxysmal sneezing, or profuse serous or sero-mucous discharges, may accompany a great variety of intra-nasal disorders. In some cases with these symptoms you may find nothing very abnormal on rhinoscopic examination. In many you will find a deflected septum, or a bony spur or ridge, projecting from the septum, and impinging upon the turbinated body. Sometimes you may find mucous polypi present. In a case I saw lately, that of a young girl *æt.* 18, there was incessant sneezing and a good deal of serous discharge from the nose. I could find nothing abnormal in the nose, but there was a large mass of adenoid growths in the nasopharynx. I removed the adenoids, and the sneezing attacks were ended.

In treating such symptoms as paroxysmal sneezing and rhinorrhœa, if nothing abnormal is present in the nose you may still get good results from applying the galvanic cautery to the inferior turbinated body. General treatment is also useful, and the drugs most likely, in my experience, to benefit are quinine, belladonna, iodide of potassium, and arsenic. When pathological conditions are present they must be treated according to their nature. As regards the treatment of hypertrophic rhinitis, if it is only slightly marked, the galvano-cautery may suffice. If it is more marked it will be necessary to remove portions of the hypertrophied mucous membrane with the snare or otherwise. In very marked cases such as this, as well as in less marked cases, when the nasal passage is very narrow, turbinotomy is the best, quickest, and most satisfactory treatment. There is no better instrument for this purpose than the turbinotome or spokeshave of Carmalt Jones. With it a clean sweep can be made of the inferior turbinated body in a few seconds. Nitrous oxide gas is the most suitable anæsthetic, and allows plenty of time for the removal of one or both bodies. I should mention that this man has been completely relieved of his symptoms since the operation.

Gonorrhœa.—R. Perchloride of mercury, 1; antipyrin, 100; distilled water, 10,000. The injection four times a day, and retained as long as possible. The addition of antipyrin prevents smarting.—Dr. VATIER.

NOTES.

The Pathology of the Diarrhœal Disorders of Childhood.—From an extended clinical and pathological study at the Kaiser and Kaiserin Friedrich Kinderkrankenhaus in Berlin, Baginsky ('Archiv für Kinderheilkunde,' Bd. xxii, H. 3—6) concludes that the diarrhœal disorders of childhood arising under the influence of high summer temperature are at first only functional in character, consisting in changes in the motor and secretory functions of the gastro-intestinal tract, with abnormal digestive action. In their further course profound anatomical alterations take place in the walls of the stomach and bowels, which may range between catarrh and necrosis of the mucous membrane. The follicular changes are processes of peculiar character and independent of the catarrhal, with which they may in the course of time be associated. They lead sometimes, in addition to superficial changes, also to ulceration. These changes are attributable not to specific bacteria, but to the ordinary saprophytic micro-organisms of the intestinal tract that assume especial virulence. Under peculiar circumstances other bacteria not ordinarily found in the intestinal tract may act as causes of diarrhœal disorders. These also induce profound anatomical changes in the walls of the bowel. The invasion of other organs by these bacteria is not unusual, but is rather relatively common with regard to the kidneys. Under these circumstances the bacteria may cause profound anatomical lesions, even to the extent of suppuration. The transmission does not usually take place through the blood-stream, the bacteria being but rarely found in the blood, and then only in small numbers. The most profound disturbances are occasioned by the fermentative products of bacterial activity, toxic or non-toxic. These are either of the nature of acids or products of albuminous degeneration, down to ammonia and its combinations, which behave as active irritants, and thus cause injury to the walls of the bowel. Further, through the blood-current and the lymph-stream they exert a degenerative influence upon other organs, especially those possessed of secretory functions, such as the liver, the kidneys, &c. Under the influence of this intoxication from the intestinal tract the resistance of

the whole organism to the invasion of other pathogenic micro-organisms is diminished, as is manifested by numerous complications.

Medical Record, July 10th, 1897.

When to Amputate in Preference to the Usual Operations for Lacerations of the Cervix Uteri.—

Thomas Addis Emmet, of New York, read a paper with this title at the meeting of the American Gynecological Society. It had been thirty-five years since he had first practised what was known as Emmet's operation for the repair of the cervix uteri. The necessity no longer existed, in this country at least, for the author of it to stand on the defensive. The judgment stood that in appropriate cases better results could be obtained through its agency than by any other operation. It was only in those cases which were exceptions to the rule, and could not be cured by preliminary local applications and Emmet's operation, that he advocated amputation by the method described in his book and again in his paper to-day. Some women had not the time or means to undergo the long preliminary treatment required for softening the cervix, &c., in the simpler operation. In others the cicatricial and diseased tissue extended so deeply into the cervix that even prolonged local and general measures (not operative) would not put the cervix in condition so that the lips could be brought together after all diseased portions had been removed. It must not be inferred, however, that he was not as strong an advocate as ever of a conservative course, that of saving the cervix whenever possible. The uterus was drawn gently to the vaginal outlet, held there steadily by an assistant; the arteries thus stretched were narrowed, and the hæmorrhage was much diminished. It was important, too, as the excavation went on for the assistant to draw successively upon the tissues to be cut, as in this way the hæmorrhage, which otherwise would be excessive, would be slight. The cervix was taken out cone-shape. One should not go out as far as seemed safe, otherwise there would be too great retraction, and possible injury of the peritoneum or bladder. As the operation proceeded, the bottom of the cavity must be kept at the vaginal outlet. To obviate closure of the uterine canal by contraction of the tissues, and leaving a cicatrising surface upon the uterine stump, he drew vaginal tissue over the

stump from opposite sides and secured it permanently upon its surface. The cervical canal was left fully open. A silver suture was passed from the required point out on the vagina inward through the stump, stopping short of the central opening; a similar one on the opposite side; others, one or more, in opposite surfaces on both sides of the cervical stump, and all were properly tightened after the uterus was first replaced in a normal position. The sutures were removed about the twentieth day. No operation gave better results in cases suitable to it.

The Charlotte Medical Journal, June, 1897.

Nasal Obstruction and the Symptoms of Cardiac Disease.—

George Roe Lockwood concludes a practical paper on nasal obstruction and the symptoms of heart disease as follows:

1. It is highly probable that patients with cardiac disease are more subject than are others to nasal obstruction.

2. Nasal obstruction occurring in a patient with cardiac disease may upset the balance of respiratory compensation and produce decided symptoms.

3. Unless care be taken these symptoms may be mistaken for those of failing compensation, and may lead to a gloomy prognosis and a faulty treatment.

4. Unless the nasal obstruction be properly relieved, and the patient allowed a sufficient quantity of good air, the arterial spasm may possibly occur, throwing an increased amount of work on the heart, already handicapped, and may become a factor in inducing dilatation. The effect of the poor quality of the blood thus supplied to the endocardium must also be taken into consideration.

5. Nasal examination made during the day may not reveal the actual obstruction, which is most apt to appear at night, when the patient is recumbent, and the circulation is in its most sluggish state. To the congestion of the posterior portion of the inferior turbinated bodies thus induced the characteristic nocturnal attacks are to be ascribed through the medium of asphyxia and arterial contraction. Nasal examination, however, usually reveals extreme vaso-motor irritability of the turbinated bodies.

6. In cases of cardiac disease, including angina and pseudo-angina pectoris, no estimate of the

patient's condition can be made, and no rational treatment can be inaugurated without a thorough examination of the patency of the upper respiratory passages.—*Therapeutic Gazette*, June, 1897.

Rough Notes on Remedies. By WILLIAM MURRAY, M.D. 2nd Edition. London: H. K. Lewis, 1897.

These notes are evidently the outcome of many years' careful observation and varied experience. The thoughtful and interesting remarks on the action of such well-known remedies as arsenic, belladonna, and mercury, most efficiently succeed in rousing the reader's attention to the fact that the action of these therapeutical agents is by no means limited by our present knowledge. The volume is deserving of careful study, especially those passages where it is, happily, pointed out that a drug should not be regarded as useless until we are assured that the fault does not arise from a want of proper application, or from lack of a proper perception of the time when to use it and the mode or method of its employment.

Notes on the more Common Diseases of the Eye.

By ROBERT W. DOYNE, F.R.C.S., Surgeon to the Oxford Eye Hospital, &c. London: H. K. Lewis. Price 2s.

In this small book of 47 pages Mr. Doyne has succeeded in given a general idea of the more common eye troubles, in explaining the principles that underlie their treatment, and in pointing out some of the practical difficulties and mistakes that may occur. Most practitioners have by them one or more of the comprehensive treatises on the science of ophthalmology, but in the ordinary course of events it is not probable that many practices furnish a sufficient number of illustrative eye cases to enable medical men to thoroughly follow their teaching; in fact, to put the matter shortly, this book is sure of a wide sphere of usefulness, because there are very many who will welcome, and we think welcome wisely, this plain, straightforward work, not only for saying what should be done, but also for emphatically indicating what should not be done; in this lies the value of this little volume. It is a difficult matter to single out any particular part of this work for special notice

because of the even quality of the excellence of the whole book; on account of the errors in treatment to which the cornea is liable, and the careful observation that should be exercised in regard to this structure while treating affections of the conjunctiva, it is certain that the author's teaching under the heading of Cornea will be widely appreciated. In writing of glaucoma Mr. Doyne truly says that "the number of patients seen with hopelessly blind eyes in consequence of wrong treatment or lack of appropriate treatment is truly appalling." This state of things should not exist, because it is preventable, and we hope that one result of the publication of this volume may be that in some future edition Mr. Doyne may have occasion to modify his estimate of the frequency with which the acute attacks of glaucoma are "mistaken for 'bilious' attacks and treated accordingly; while the eye, which is the exciting cause, and not the liver, is allowed to go blind."

Hypodermic Tabloids of Potassium Permanganate, 2 grs. (B. W. & Co.).—The ready solubility of these tabloids furnishes the practitioner with a handy means of immediate treatment in cases of opium poisoning. Hypodermic Tabloids of Ergotinine Citrate, $\frac{1}{100}$ gr., with Strychnine Sulphate, $\frac{1}{20}$ gr. (B. W. & Co.).—The therapeutical effect of these tabloids is increased by the strychnine salt, and the combination is a distinct advantage. According to recent reports these tabloids have been found beneficial in uterine hæmorrhages and allied disorders; their action also is obtained when administered by the mouth, and the accurate adjustment of the dosage renders this form of preparation a very valuable remedial agent.

New Books.—Messrs. Longmans & Co. announce the following as in the press:

The Diseases of the Lungs, by James Kingston Fowler, M.A., M.D., F.R.C.P., and Rickman J. Godlee, B.A. Lond., M.B., F.R.C.S. *A Manual of Midwifery*, by William Radford Dakin, M.D., F.R.C.P. *The Treatment of Wounds in War*, by Surgeon Lieut.-Col. W. B. Stevenson, Army Medical School, Netley. *Surgical Pathology and Principles*, by J. Jackson Clarke, M.B. Lond., F.R.C.S. *Lectures on Animal Electricity*, delivered at the Royal Institute of Great Britain; by Augustus D. Waller, M.D., F.R.S.

THE CLINICAL JOURNAL.

WEDNESDAY, AUGUST 4, 1897.

A CLINICAL LECTURE ON MENTAL DEFICIENCY IN CHILDREN.

ILLUSTRATED BY CASES.

Delivered at the West End Hospital for Diseases of the Nervous System, March 16th, 1897.

By **FLETCHER BEACH, M.B., F.R.C.P.,**

Physician to the Hospital; formerly Medical Superintendent of the Darenth Schools for Imbecile Children.

GENTLEMEN, — Mental defectiveness depends usually upon imperfect development, or disease of the nervous system, dating from birth or early infancy. It must, however, not be forgotten that besides the mental state which exists in each case, certain physical characteristics are present, such as a slouching gait, want of co-ordinating power of the body and limbs, a wandering eye, inertness or too great restlessness, abnormal head, and signs of the scrofulous cachexia. The presence of these and other conditions in association with fixed mental conditions enables us to divide the cases before us into definite groups.

CLASSIFICATION: There are various systems, but the one which I have adopted is very simple. Cases are divided into congenital, *i. e.* occurring at the time of birth; and non-congenital, in which the defect appears after birth.

CONGENITAL: 1. Simple Congenital. 2. Microcephalic. 3. Hydrocephalic. 4. Scaphocephalic. 5. Paralytic. 6. Cretinism, sporadic and endemic.

NON-CONGENITAL: 1. Eclampsic. 2. Epileptic. 3. Hydrocephalic. 4. Paralytic. 5. Inflammatory, sub-class Hypertrophic. 6. Traumatic.

Simple Congenital.—Under the first class I include children born without any obvious defect or abnormality of the skull and limbs. This classification includes some cases of a very low and others of a high type. In the low class cases, as in the children I now show you, you will often notice an animal expression—thick lips,

pug nose, large, coarse, outstanding ears, defective palpebral fissures, broad, thick, depressed bridge of the nose, a hairy or narrow forehead, and a heavy underhung jaw. In the higher classes the facial appearance and expression is most marked. All have a fairly intelligent look, and the expression on the whole is pleasing. All, both boys and girls, make good progress under proper education, and the boys learn trades, while the girls are usefully employed in domestic work.

Mongol type.—Children of this type are so called from the similarity in their facial appearance to the Chinese. They have obliquity of the orbits, so that instead of these being on the level as ours are, they have a tendency to be directed upward and outward. Besides this, these children usually have broad features, rounded pinnæ to the ears, hypertrophied papillæ of the tongue, which often presents transverse fissures, roughness of the skin, and hands and feet short and broad. Often they are stunted in stature. As a rule they are very imitative, and see the comic side of everything. Their circulation is feeble, and they are very liable to bronchitis. Many are born of mothers who have suffered continued ill-health or depression during pregnancy, and they are, if we may say so, unfinished children. In many cases they are the last born of a long family, and are born of parents far advanced in life.

I should have mentioned that many of this simple congenital class, as well as of the classes which I shall afterwards relate, have a high palate, which may be V-shaped, too narrow from side to side, or otherwise deformed, or which is called neurotic in shape. This last palate, according to Dr. Clouston, who first described it, is one midway between the normal palate and the V-shaped palate, and was so called because it was frequently found in persons of a nervous temperament, who were liable to hysteria, neuralgia, and migraine. I have seen many of these palates in the children at this hospital. It has a more Gothic arch than the normal or typical palate, and the alveoli run parallel for a greater distance than in the V-shaped palate. Some years ago, I examined 700 feeble-

minded children, and found that of every 100 about 60 had neurotic palates, and 28 had V-shaped or deformed palates. It was formerly held that all children who had high palates, or V-shaped palates were necessarily congenital cases, but later inquirers have contested this point. Another form of palate sometimes met with is the long flat palate with projecting teeth; this is sometimes connected with a short under jaw, and then the upper jaw projects over the lower one.

Microcephalic.—These are children whose heads are smaller than normal, measuring in circumference from $15\frac{1}{2}$ to $17\frac{1}{2}$ or 18 inches. I have seen one child, a girl aged 12 years, whose head measured only twelve inches in circumference, and after death her brain was found to weigh only seven ounces. This girl led, for the most part, a vegetative life, but she learnt to recognise those around her, became cleanly in her habits, and made an attempt at articulation. The forehead usually is narrow and slopes backwards, and correspondingly we find after death the brain very small in this position. The occiput is also not properly developed. The features are frequently shapely, the eyes large, and the nose aquiline. In some cases the children have a bird-like aspect, in others the face is large in proportion to the cranium. In those whose heads measure from $17\frac{1}{2}$ to $18\frac{1}{2}$ inches, not only are the features shapely, but the children will be found to be imitative, restless, and fond of music, but they have little power of attention; this faculty has to be developed, and in proportion to its development will be the amount of improvement to be expected. These higher classes do make some improvement: as a rule they learn to speak, read simple words, and match a few colours. The cerebral convolutions in these cases are usually very simple, and, as a consequence of the defect of development in the occipital lobes, the cerebellum is left to a great extent uncovered. At one time it was thought that the small size of the brain was owing to the premature synostosis of the cranial sutures compressing the brain and hindering its growth. It is now known that there was no foundation for this opinion, and consequently craniectomy, so far as microcephalic mental deficiency is concerned, is not now required. In those cases in which it was performed very little improvement, or none at all, was the result, and indeed we could not expect

much from it, for the microcephalic head is a sign of a vice of nutrition which affects the trophic functions of the whole nervous system. In some cases the brain ceases to grow after the fourth or fifth month, and it is obvious that no operation can alter this condition.

Hydrocephalic.—These children have very large heads. I now refer to children who were hydrocephalic at the time of birth. According to Meynert, in the congenital form the lateral ventricles are extended in their long diameter, while in acquired hydrocephalus the ventricles are increased in their transverse and vertical diameters. The treatment is just the same in both cases. All cases of hydrocephalus do not, of course, become weak-minded. Many recover, but there are some who do not die, and do not recover, but become weak-minded. The head approaches the globular form, and the antero-posterior and transverse diameters are nearly the same. The widest circumference is often at the temples. Children of this class can often read and write simple words, repeat easy multiplication tables, and recognise a few colours. Of course, everything depends upon the amount of serum poured out and the amount of brain left undamaged. If, as is sometimes the case, the amount poured out is large, there will not be much brain left to train. In one case which was under my care, the lateral ventricles were found to contain thirty-six ounces, or nearly two pints of fluid. Above them the brain substance was very thin, being not much more than a quarter of an inch in thickness on the convex surface. The convolutions, too, were very coarse, some of them measuring as much as one inch in width, so that there was deficiency in quality as well as in quantity. In this case, of course, no improvement could be effected. If you examine a section of brain, not only of this class but of others, under the microscope, you will frequently find that the cells are rounded or pear-like in shape, and that they often have few processes. The apical process is the most persistent. Often the contents of the cell are commencing to degenerate or have undergone degeneration.

Scaphocephalic.—These are cases which have a keel-shaped head, but whether produced by difficult labour or not I have been unable to ascertain. These cases are not very common, but I have seen

three or four. One of the most marked cases I had under my care was that of a boy aged 11 years, whose head measured $22\frac{1}{2}$ inches in circumference, $12\frac{1}{2}$ inches transversely, and $16\frac{1}{2}$ inches in the antero-posterior direction, so that there were four inches difference between the last two measurements. He spoke indistinctly, and during the time he was under care made little progress.

Of course, everything depends upon the quality of the brain; if this is defective there will be little chance of improvement. There are sometimes other shapes met with, such as the head distorted in shape, the asymmetrical head, and the sugar-loafed head. Too much dependence must not be placed on the shape of the head; it is the shape of the head in combination with other factors which goes to make up the mentally defective child.

Paralytic.—I now refer to cases paralysed at the time of birth. Paralysis may be due to some inflammation of the membranes in utero or be due to pressure at the time of birth—cases of birth-palsy, in fact. Generally these children make good progress mentally, if there are no fits and the injury to the brain is not great in amount; but physically, that is, with regard to paralysed limbs, there is little improvement. One interesting case, which some years ago I had under my care, was due apparently to the mother when pregnant falling with great violence and striking her side against a wall. The boy was born with paralysis of the right side. In that case there was flattening of the cranium on the side opposite to the paralysis. Under appropriate treatment he gained a considerable amount of power in the right hand, which was more affected than the leg, and coincidentally with the improvement the flattening of the skull became less noticeable.

Sporadic cretinism.—Children of this class are very stunted in growth. The head is usually large, flat at the top, and spread out at the sides, and the hair is often sparse, coarse, and dry, like horsehair. The nose is pug-shaped, the cheeks full and flabby, the mouth large, and the lips thick and often slightly apart. The neck is short and thick, and there is no sign of a thyroid gland. Well-developed fatty swellings are seen in most cases on each side of the neck, and sometimes in the arm-pits. The abdomen is large, distended, and contains a quantity of subcutaneous fat; frequently there are umbilical and sometimes

inguinal herniæ. The arms and legs are short and curved, and the hands and feet in some cases, though small, are broad, thick, blue, and usually dry and scaly. The skin is dry and rough, owing to the almost entire absence of perspiration; on the face it is yellow and waxy, giving rise to a sallow colour. The pulse is small and feeble, and the temperature subnormal, so that there is great susceptibility to cold. The voice is rough, harsh, hoarse or squeaky; the gait is clumsy and waddling, and often there is repugnance to making any movement. Speech is generally limited to a few words, often monosyllabic in character, and consisting of "Yes" and "No." They are good-natured, placid in temperament, and fond of those who attend upon them.

That is the description of the cretin who has undergone no treatment. In those who have undergone treatment, as in these five children, certain differences will be noted. The fatty swellings on each side of the neck disappear, the prominent abdomen subsides, and the umbilical hernia becomes lessened or entirely disappears. The skin of the body and extremities desquamates, and is no longer dry and scaly, and the temperature rises. As a rule the expression becomes more intelligent. Patients begin to grow, and their weight is lessened. Cutaneous sensibility approaches the normal condition. The patients I have under treatment are taking thyroid tabloids—the young patients five grains daily, and the older patients ten grains daily. Treatment has to be kept up during the patient's lifetime, otherwise they lapse into their old condition. The treatment should be carefully watched, and if the patient loses weight too rapidly, or the temperature rises above the normal, if nervous troubles of any kind and tachycardia make their appearance, the treatment must be stopped for a time, or the dose reduced.

The cause of the disease is, as is well known, the absence or disease of the thyroid gland.

Endemic cretinism is not met with much in England, but is seen in Switzerland, and is due to patients living in valleys shut in by high mountains, so that the sun penetrates into the valley only for an hour or two a day. Some years ago Dr. Guggenbuhl removed a number of these patients from the valleys, and built a home for them on the top of a mountain. The effect of the

change was surprising. The goitres which these patients have, to a great extent disappeared, and the patients became more intelligent.

Passing now to the *non-congenital* cases, we shall notice that many of the signs which distinguish the congenital class are absent. In fact, children belonging to the non-congenital class often have a bright expression, a good-shaped head, and well-made limbs. They can generally walk and run well, and if the faculty of attention is well developed they learn with not much difficulty. Unfortunately, many of them are so mobile, so constantly on the move, and the eye is so restless that the attention cannot be fixed.

Eclampsic.—These are cases in which convulsions have come on soon after birth, continued for some years and then ceased, but have so altered the structure of the brain that the child has become weak-minded. In many of the cases which have come under my notice there has been a family history of intemperance, or a history of insanity or epilepsy in the parents, and the child has been handicapped as it were in the race of life, and had less chance of recovery from the fits without loss of intellect.

In a case which was under my care at Darenth, the child appeared mentally sound at birth, and was a lively child up to two years of age. She had convulsions when cutting her teeth, and they continued until she was five years old. After the fits ceased she became excitable, and afterwards very quiet. The mischief by that time was done. Both her paternal uncle and grandfather were insane, so that she was born in all probability with an unstable brain, which was upset by the convulsions and fits. She made very little progress under treatment.

Epileptic.—This class includes some cases in which, with cessation of fits, the greatest improvement takes place; others in which the patient remains *in statu quo*, and others still in which the patient goes on from bad to worse, the fits becoming more frequent and severe, and the result is utter dementia. The children I now show you present all these conditions.

Hydrocephalic.—The children I spoke to you of some time ago were hydrocephalic and imbecile at the time of birth. I now go on to describe the cases which, though hydrocephalic at the time of

birth, did not become weak-minded until some time afterwards.

This patient, whose photograph I show you, was aged 18 on admission to Darenth, and was dull and listless. His father and paternal grandfather died of apoplexy, and two paternal uncles were insane. In addition, all the father's side of the family were excitable, and there is a history of phthisis on the mother's side. This illustrates the fact that most commonly several well-known causes act together to produce the result. Rarely is only one cause known to do so. When eleven years old he screamed and became very excitable, and there is no doubt he had a fit, because it was noticed afterwards that he became paralysed on the right side. He remained in this condition for six months, and then gradually recovered. There was no paralysis when he was admitted, though he was weak in both legs. As he grew up he was noticed to be weaker, and to become dull. He had every chance of a good education, but could not learn. When admitted he could only count to six, and though he went to school in the asylum for three and a half years, he only learnt to read and write a few letters, repeat easy multiplication tables, and recognise a few colours. He deteriorated gradually, became very weak, and died of exhaustion from diarrhoea. He had no fits while in the asylum, so there is no doubt that the gradual deterioration was due to an increasing quantity of fluid pressing on the brain tissue.

Paralytic.—I now refer to children who become paralysed after birth from repeated fits (I have seen such cases), cerebral apoplexy, or atrophy of the brain. In these cases, as in those born paralysed, there is mental improvement under training if the patient is not subject to fits, but the paralysed limbs make little progress towards recovery. The time for improvement of the limbs has passed before the children come under treatment.

Schroeder Van der Kolk has collected several cases in which there was found shortening and atrophy of the limbs on one side, and atrophy on the opposite side of the brain. In the majority of these more or less weak-mindedness existed, but this was not always so. I have seen several such cases myself.

He very truly says, "Everything depends upon the more or less healthy state of one hemisphere of the brain. If, as from the nature of the case

seldom occurs, the inflammation and affection of the pia mater has not extended to this hemisphere, if the grey matter under the cerebral convolutions has here continued perfectly sound, there is no reason why this remaining hemisphere should not be able to act without impediment in the exercise of those functions which are necessary to our mental powers, just as one eye sees as sharply though the other be lost. But when the grey matter is injured in both hemispheres, particularly anteriorly, disturbance of the intellectual faculties will be inevitable."

Inflammatory.—By this term I mean weak-mindedness which has come on after some illness, such as measles, enteric fever, whooping-cough, &c., as a result or complication of which there may be inflammation of the brain or membranes—not sufficiently grave to be fatal, but serious enough to cause mental impairment.

"The amount of damage to the intellectual powers," as Dr. Ireland says, "must be mainly dependent upon the intensity of the morbid process." This, unfortunately, we have seldom a direct opportunity of measuring, as the patient does not come under the notice of medical men connected with training institutions until long after the disease has passed away.

If one of the diseases above mentioned should occur in a child previously disposed to weak-mindedness, by being born of parents whose family history shows the existence of marked neuroses, there would be the greater likelihood of weak-mindedness following.

Hypertrophic.—I have placed this class under that of the inflammatory, because the post-mortem appearances of patients who died of this disease at Darenth Schools showed that there was or had been chronic inflammation of the brain. The history of a case is much as follows:

I take it from an actual case that was under my care. The boy was born apparently perfectly healthy, but when teething had a fit, and has had them ever since. (This occurrence of fits is not always present.) He was always dull and sleepy, and as a child used to "bob" his head forwards, *i. e.* let it go forward with a jerk. The head was large when he was born, but the projections on his forehead have since come on. He is a fairly well-grown boy for his age, but has a very vacant look. The head is large, square in shape, and there are well-

marked frontal prominences. (These may not always be present.) He complains at times of headache, and points to the right temporo-parietal region when asked where the pain is situated. There is a slight depression, the size of a sixpence, in the region of the anterior fontanelle. He walks slowly and totteringly, hanging his head forward slightly. He cannot stand long at a time; soon he begins to lean forward, and would fall if not supported. He went to school in the Asylum regularly, but made no progress. Questions were answered slowly, and there was a distinct pause before the reply commenced. He suffered much from headache, and gradually deteriorated. Towards the last he became weaker on his legs, and fell about more. Finally he had a number of severe epileptic fits, and died exhausted. On removing the calvaria at the autopsy, the brain sprang upward as if relieved from pressure. It weighed fifty-three ounces, was hard, and cut like cheese. The convolutions were simple in arrangement.

According to Andral there are two periods in the disease: in the first, the chronic stage, the symptoms are slight; in the second, unless the patient has been carried off previously by the intervention of some other disease, those characterising an acute affection appear, and the patient dies of compression of the brain or acute hydrocephalus. Out of twelve cases which were under my care at Darenth Schools eight died—four from convulsions, two in a comatose condition, and two from diarrhoea and bronchitis. The brain, though heavy, is not the largest I have seen; in another patient, aged fifteen, the brain weighed 62 ounces. This affection is not so common as hydrocephalus, but is often mistaken for it, and it is important that you should know how to distinguish the one from the other. I had the opportunity of seeing several cases when at Darenth Schools, and I found that the diagnosis of hypertrophy of the brain from chronic hydrocephalus rested chiefly on the history of the case, and the size and form of the head.

Diagnosis.—1. In hypertrophy the head as a rule does not attain so large a size as in chronic hydrocephalus.

2. In hydrocephalus the increase in the size of the head is most marked at the temples; in hypertrophy above the superciliary ridges.

3. In hypertrophy the head approaches the square in shape; in hydrocephalus it is rounded.

4. In hydrocephalus there is often elasticity over the late closed fontanelle; in hypertrophy there is none, and there is often a depression in that situation.

5. In hydrocephalus the distance between the eyes is increased; in hypertrophy this is not the case.

Traumatic.—In this class are included cases in which, from a fall or blow on the head, the patient becomes weak-minded. Under this heading also come cases resulting from injury to the head, caused by narrowness of the pelvis and prolonged labour. The degree and nature of the mental defect so produced must vary with the amount of the destruction of the nervous tissue. As Ireland observes, "Sometimes the injury to the mental power is permanent, sometimes it disappears more or less slowly; in some cases a trifling injury causes grave disorder, in other cases what appears to be a great injury leaves no visible effects behind." Hereditary predisposition has much to do with this.

The more I see of these cases the more I am impressed by the importance of hereditary predisposition. In fact, the prognosis of the case depends to a great extent on the presence or absence of hereditary predisposition, or rather on the nature of the predisposition.

Time will not allow of my entering fully into the ETIOLOGY of the affection, so I can only touch briefly upon it.

A few years ago my friend Dr. Shuttleworth and I made an examination into about 2,400 cases, of which we had obtained histories, and we found of the cases *acting before birth* (without giving decimal points), phthisis was accountable for 28 per cent., insanity and epilepsy for 21 per cent., and epilepsy and other neuroses for 20 per cent. Intemperance accounted for 16 per cent., but syphilis was acknowledged in only 1.17 per cent. This percentage of syphilis agrees very closely with other observers. What syphilis in the parents often does is to cause a gradual breakdown of the nervous and mental system of the child at puberty.

Consanguinity only accounted for 4 per cent., even when the consanguinity of the grandparents was taken into account, so that it does not play

such an important part in the production of mental deficiency as is generally supposed. Even in those cases where consanguinity was present there were other hereditary tendencies of themselves quite sufficient to produce the affection without the presence of consanguinity.

Abnormal conditions of the mother during pregnancy, both physical—such as a blow, or mental—such as worry and anxiety or fright, accounted for 29 per cent. Of *causes acting at birth*, premature birth only accounted for about 4 per cent., while prolonged parturition was responsible for 14 per cent. On the other hand, forceps delivery only accounted for 3 per cent. This is in accordance with what I have always taught, that it is better to put on the forceps and deliver the mother than to allow her to drag on a weary confinement. Not only does prolonged compression of the head result in asphyxia, but a number of the children when born are in a helpless condition, some having lost the use of their legs, others becoming subject to convulsions; moreover, the head is often crushed, distorted, or otherwise injured. When death ensues early, meningeal hæmorrhage is formed on the convexity of the brain, thickest over the central zone, and in some cases there is actual cortical laceration. On the other hand, in the cases which have been delivered by forceps, only a few are helpless or paralysed. Of *causes acting after birth*, infantile convulsions account for no less than 27 per cent., while epilepsy and other cerebral affections for only 8 per cent. Injury to the head from a fall or blow was responsible for 6 per cent., while fright or shock only produced 119 cases, or 3 per cent. Febrile illnesses, such as scarlatina, whooping-cough, measles, enteric fever, and smallpox were accountable for nearly 6 per cent., while over-pressure in school was only given as 0.16 per cent. There are other slight causes, but those I have given are the most important.

Coming now to the DIAGNOSIS. If we are called to a case which occurs in infancy, we should observe the shape of the head, which may be obliquely deformed, boat-shaped, elevated, elongated, too small (microcephalic), or too large, owing to the presence of chronic hydrocephalus, or hypertrophy of the brain. Next the size, as heads below 17 or even 18 inches in circumference rarely show signs of much intelligence; then notice

whether the child is able to support it, or whether it hangs back motionless. Also, whether the muscles of the spine are able to support the body, or whether the child has to be supported by the hand and arm of the mother. Notice whether the limbs are flaccid, or, on the other hand, contracted. Whether there is any difficulty in swallowing the milk drawn from the breast, whether the child is capable of grasping one's hand; whether any notice is taken of passing objects, and whether the child can follow them with his eyes. Whether notice is taken of sound, and whether there is any voice or attempt to speak. The presence or absence of strabismus or nystagmus should also be noted, as well as the distance between the eyes, which are too near in microcephalic, and too widely apart in hydrocephalic cases. The position of the eyes, whether obliquely placed or not, should be carefully examined, as the former condition is met with in Mongolian patients. The integument around the eyes should be examined for epicanthic or semilunar folds of skin at the inner canthi, and the position of the ears, whether implanted too far back or not should be noted.

If you find that the child has too small or too large a head, which is also badly shaped, that he cannot support his head, or sit up in his nurse's lap; that his limbs are flaccid and never put in movement, or that they are contracted; that he takes no notice of passing objects, or does not follow them with his eyes; that he takes no notice of sound, and does not attempt to speak, you may be sure that you have to do with a mentally deficient infant. The additional presence of strabismus; or nystagmus, obliquely placed orbits, epicanthic folds and large ears implanted too far back will aid the diagnosis. Later on, comparison is made of the child's intellect with that of others of the same age. You should notice whether the child has good power of observation, imitation, and especially of attention, as without the latter little improvement in the mental condition can be expected; whether his memory is good, and whether he can read and write, or has any knowledge of arithmetic. The facial aspect should be observed as there may be want of symmetry, or coarse, heavy, flat features, thick lips, defective palpebral fissures, hairy forehead, large, coarse, outstanding ears, implanted too far back, and a prognathus heavy, underhung jaw. Many of these conditions

are found in cases of low type. You should notice whether the fontanelles are open or closed; the shape of the palate, which is often highly arched; the grasping power of the hand, which is occasionally weak; the presence or absence of automatic, purposeless movements, contractions or spastic rigidity; whether there is any flow of saliva from the mouth; the state of the circulation, which is usually feeble; the amount of development of the senses; whether there is sluggishness of movement or too great restlessness; the presence or absence of speech, of will, foresight, prudence, and perseverance, which is usually absent in these cases; and the presence or absence of epilepsy.

The congenital cases can usually be distinguished by a small or badly-shaped head, a shelving forehead, heavy, coarse, flat features, a V-shaped palate, large ears implanted too far back, indistinct speech, and in low class children, a flow of saliva from the mouth, and impassive, sluggish, or automatic movements. The Mongolian and Cretinoid varieties will be distinguished by the characteristics previously mentioned. The non-congenital cases often have a bright expression, a head of good shape, well-made limbs, activity of movement, and fairly good speech. Many of these children, however, cannot learn much, for the eye is perpetually wandering and the attention cannot be fixed.

As regards PROGNOSIS. Contrary to what may be expected, as a rule, the prognosis is better in congenital than in non-congenital cases. In the first class we simply have to do with a defectively-developed brain, while in the latter there is frequently disease of the brain, often irremediable. There are a few cases in which the traumatic damage may be slight, or in which the post-febrile lesion is not great, or in which the epileptic fits have been slight, and have yielded to treatment. All these will improve, but, as a general rule, the prognosis is, as I have stated, better in the congenital than in the non-congenital cases.

As regards the various types. With respect to the *microcephalic*, this will vary according to the size of the head. With heads under 18 inches in circumference little can be done, but from 18 to 19 or 19½ inches there is hope of improvement under training, especially in useful industrial work. An occupation not requiring headwork is what they are best fitted for.

As regards the *hydrocephalic*. After the acute symptoms have subsided much may be done, but the amount of improvement depends upon the amount of serum thrown out and the damage that the brain has undergone. In the case I previously mentioned, where after death only a quarter of an inch remained on the convex surface, little could be expected, but other cases under my care learnt to read, write, and do simple sums.

The *Mongol* type of child, as I said before, is very imitative—he sees the comic side of everything, and is fond of music, so that he soon learns drill and dancing. There are various gradations of this class of child, and the higher classes learn to read, write, draw, but cannot do much at arithmetic.

The *paralytic* cases, as I think I mentioned, improve mentally but not much physically, although the growth of the muscles, if some of the nervous strands remain undamaged, may be aided by massage and galvanism.

The *eclamptic* cases do not improve, the mischief has been done before the children come under training. As regards the *epileptic*, if the fits have not lasted long and are amenable to treatment, as I said before, there will be improvement. In many cases the mental condition appears to remain in the same state for years, while others pass rapidly into a state of dementia. *Inflammatory* or post-febrile cases do not improve much if the brain has sustained much damage; in some cases there is atrophy of the brain consequent on meningeal thickening, in others the arrest of development may be slight, and then improvement may take place. *Hypertrophic* cases do not improve; all that we can hope to do with these is to keep the patient in as healthy a state as possible, and treat any active symptoms which may arise.

Traumatic cases improve if the damage done is not great, but if it is, no improvement can be hoped for. The *Cretinoid* cases I have already mentioned improve, as long as the thyroid gland is administered, but even then no great mental improvement can be hoped for.

In *syphilitic* cases the prognosis is always grave, as mental enfeeblement often sets in at the age of the second dentition. In some cases there is progressive dementia with epileptic seizures, and the child dies in a few years after the onset of the disease.

THE TREATMENT* I can only just allude to, as this would require a lecture of itself. It consists of a judicious combination of *hygienic*, medical, physical, moral, and intellectual treatment. As there are physical imperfections as well as mental deficiencies, it is necessary to improve the former if we would ameliorate the latter condition. The want of co-ordinating power in the muscles has to be improved by appropriate remedies. The muscular treatment being strengthened, the hands have less difficulty in performing any simple act; locomotion is improved, the eyes wander less, and listlessness and inertness to a great extent disappear. On the other hand, the restlessness of the excitable class must be soothed by appropriate remedies. The moral treatment should go on side by side with the physical and intellectual training. Ideas of justice, duty, self-reliance, prudence, forethought, and perseverance have to be inculcated. Obedience has to be taught and efforts made to impart good temper and affection. With regard to the intellectual training, we must remember that mentally defective children have to be taught ideas and motives which ordinary children pick up for themselves. The latter are endowed with the full use of their senses, while in the former these are badly developed and have to be trained. It is usual to commence by educating the senses, and as the tactile function is the most important, we begin by educating the sense of touch. Then follows the education of sight, hearing, taste, and smell. In every case it is necessary to proceed from the simple to the complex, teaching ideas by the use of concrete forms, and not by abstract notions. Speech has to be educated by means of a speech-drill.

The senses having been educated and the speech improved, we advance to higher branches of learning, such as reading by the use of "work-cards," writing, arithmetic, drawing, elementary geography, &c. When good progress has been made education in Sloyd work, carpentering, and gardening for the boys, and sewing and domestic work for girls, will be found useful; alternating the industrial with the purely intellectual training.

Some *practical hints* may be found useful.

* Those who are interested in the treatment will find a fuller description in a little book by the author, 'The Treatment and Education of Mentally Feeble Children,' Churchill, 1895.

Firstly, the shape of the head and the aspect of the face cannot be relied upon as the only means of prognosis. The brightest-looking children are often the most volatile, and their attention is not easily gained; on the other hand, those who are dull-looking will frequently take an interest in their work, and make considerable progress.

Secondly, each case needs to be carefully studied and treated; some learn more by ear, others by sight, and the education has to be adapted to the requirements of each. It should be especially remembered that parrot-knowledge is of no use.

Thirdly, the children should be removed from home and placed in a suitable institution, where they can be carefully educated and trained. Home instruction is of little use; if the child is removed to an institution in which he is on the same 'mental plane' as the others, the spirit of emulation is aroused; he can join in the amusements and games, and his life becomes joyous and bright.

Lastly, the training, both mental and physical, should be commenced as early as possible; for the older the child is when the training begins, the less chance there is of ultimate improvement and recovery. Many mothers have been misled by the idea that at the age of seven or fourteen there will be an abrupt change from mental enfeeblement to mental brightness. From the nature of the case we should consider this result impossible, and experience teaches that at these ages sudden changes for the better do not occur.

A CLINICAL LECTURE

ON SOME CASES OF

SUPRA-PUBIC LITHOTOMY.

Delivered at St. George's Hospital, June 15th, 1897, by

WILLIAM H. BENNETT, F.R.C.S.Eng.,

Surgeon and Lecturer on Clinical Surgery at the Hospital;

Examiner in Surgery, University of Cambridge;

Member of Court of Examiners,

Royal College of Surgeons.

GENTLEMEN,—I propose to consider to-day some cases of stone in the bladder which have been under my care in the hospital comparatively

recently, and which I treated by supra-pubic lithotomy. I have two main reasons for calling your attention to these cases: (1) the intrinsic interest of the cases themselves, and (2) the treatment which was adopted—that is to say, supra-pubic cystotomy. There is a tendency among a certain number of surgeons to speak of cutting operations for stone in the bladder as proceedings which should soon almost cease to be heard of, excepting in out-of-the-way cases. I am one of those persons who do not think that this will be quite the case; I do not think that lithotripsy will supplant these cutting operations to the extent which some appear to think, nor do I believe it has so much to commend it for universal use as some people seem to fancy.

The first of the cases to relate is that of a little boy whom some of you may remember to have seen in the Wellington ward. He was only $2\frac{1}{2}$ years of age. He had the ordinary symptoms which children suffer from when they have stone in the bladder; that is to say, he was restless, was constantly straining to pass water, and was continually pulling at his prepuce—so much so that he had made it quite long. Upon sounding the boy's bladder, a stone was readily felt. He was a very sickly child, and rickety, with a very large forehead; in fact, he was a most unhealthy little person. I cut him above the pubes, removed a stone about the size of the end of one's thumb, and sewed up the bladder wound with catgut. The incision healed immediately, there was no leakage from the bladder wound, and the little fellow was well in a very short time, having had no subsequent pain or trouble of any kind. That, then, is a case of supra-pubic lithotomy in a child only $2\frac{1}{2}$ years old. The reason for my selection of the supra-pubic method in this case I will explain presently.

The next case I wish to mention to you is that of an oldish man—a different class of case altogether. His age was 60, and he was in Belgrave ward. He had all the ordinary symptoms of stone, from which he had suffered for three or four years. He had used a catheter for a longer time than that, because he was the subject of an enlarged prostate. On coming to the hospital he complained mostly of the occasional passage of a little blood and thick sediment in his urine; sometimes he passed blood in the urine during defæcation. I sounded him and

found a stone situated quite deeply behind the prostate. The stone, as I could feel it in the bladder, was apparently small and fixed; but on passing a finger into the rectum, which I took especial care to do because he complained of pain on defæcation, I found that projecting back into the bowel was a mass larger than the prostate, and somewhat above it. The mass was of stony hardness, and I could by pressure make it impinge upon the sound which was in the bladder. I concluded, therefore, that the case was one of a large encysted stone. Only a portion of the stone was in the bladder, the main portion of it lay in an almost complete sac. In this case I did not consider the question of lithotripsy at all. I did a supra-pubic lithotomy and found the condition I expected. There was a good deal of trouble in getting the stone out of the cyst as such a small piece of it projected into the bladder, about four-fifths of the calculus being firmly imbedded in the pouch. I sewed up the bladder wound in the same way as I closed that of the little child's, but, as often happens in older people, the wound did not unite immediately. There was some leakage of urine for two or three weeks before the wound healed, but finally everything closed and the patient recovered completely; when I saw him last he remained quite healthy. These two cases afford a good contrast, one being in a very young subject, the other in advancing life.

The next case is one of a different kind. The patient was a young man aged 24, who was in Fitzwilliam ward. He had been suffering occasionally from discomfort after passing his water; once or twice he had passed some blood, and his urine was so thick at times, and the scalding was often so bad, that he went to the Lock Hospital under the impression that he was suffering from gonorrhœa. He there saw Mr. Ward, who, seeing he had no gonorrhœa and that he had some symptoms pointing to the existence of stone in his bladder, kindly sent him to me. On sounding him I found the stone without any trouble; it was small, and moved about the bladder freely. After the first sounding I determined to crush this stone, but on sounding the bladder again I could not find the stone at all. I passed my finger into the rectum, which sometimes helps one to feel the stone, but could detect nothing in the

bladder of the nature of stone. I sounded him the third time, but again could not find the stone, although every conceivable position of the patient was tried; the bladder seemed quite free of any foreign body. Still the discharge of pus continued, and although there was no blood there was always great pain after micturition. On sounding a fourth time, just as the sound passed into the bladder I fancied I felt a stone, but I could not feel it afterwards. Indeed, in the four soundings I only thoroughly felt the stone once. Therefore it occurred to me that although I was certain of the existence of a stone, I should have a difficulty in crushing it as it was so difficult to find. So I made up my mind to perform lithotomy, and again I chose the supra-pubic method. On opening the bladder the reason of my difficulty in detecting the stone was perfectly apparent. For some short time, even with my finger in the viscus, I could find no stone, and for a moment I thought I had cut into an empty bladder after all. But on feeling about very carefully, I detected on the left side of the prostate something a little rough, and on moving my finger over this gently, I found I could pass it into a little pouch, in which there were two small stones. One of these stones, under certain circumstances, was no doubt in the habit of getting into the bladder, and then was easily accessible to the end of the sound. The stone was in the bladder the first time I felt it, and on the other occasions when I sounded the patient it had slipped back into the pouch. It was this evasive character of the stone which caused me chiefly to decide to perform supra-pubic cystotomy, in spite of the small size of the calculus. I had in my mind the possibility of there being a "wandering" stone, and my supposition turned out to be right. The result showed pretty clearly that my decision not to attempt the crushing operation was the correct one.

The next case is a very uncommon one. The patient was a woman æt. 40, who was in the Princess ward. She had suffered from all sorts of pain and distress about her bladder region for many years. She had been told by more than one practitioner that she had probably displacement of the womb. Latterly she had passed blood, and sometimes she was seized with "cramp in the bladder," the pain at times being so acute

that she was said to be in danger of going out of her mind. On many occasions some blood had been squeezed out of the urethra at the end of micturition. It therefore appeared that the case was probably one of stone in the bladder. Upon sounding the bladder in the usual way, a stone was at once hit upon. The stone was quite fixed, and appeared to be encysted in the anterior aspect of the bladder on the right side. Two days after the sounding I received an urgent summons to see the patient, on account of a sudden onset of most acute pain which was quite uncontrollable. On arriving at the hospital I found her in a distressing condition. The stone had become impacted in the neck of the bladder. I passed a sound down her meatus, and just at the end it was arrested by the stone. Seeing the extremely acute pain, and remembering the amount of blood she had passed from time to time, as well as the offensive condition of her urine, I did not attempt to crush, but decided to cut for the stone. I therefore made an incision above the pubes, and found a stone loose in the bladder rather larger than a pigeon's egg. I also found a condition which is very uncommon, and which accounted for the extreme severity of the symptoms. On the right side of the bladder, towards the front, there was a considerable amount of malignant disease, the centre of which contained a cavity of about the same size as the stone. It is conceivable, and in fact I think probable, that at one time the stone in this particular instance had been contained in a cyst in the bladder wall, in which malignant disease had developed. In the progress of the malignant disease the stone must have been loosened, and finally dropped out into the bladder. This explanation would account for the stone which I felt on passing the sound being fixed, although when I operated it was certainly lying loose in the cavity of the bladder.

In each of the four cases which I have narrated there was a very good and definite reason for preferring a cutting to a crushing operation. In the first case the patient was so sickly and wretched, and the urethra was so small that even if I had thought a crushing operation possible, I should have hesitated about passing an instrument down the urethra sufficiently large to enable me to evacuate the stone if I could have crushed it. With persons who are used to performing lithotri-

the crushing of stones in young children is not a difficult matter, but in a child so small as this I think one should hesitate before passing an instrument sufficiently large to allow of proper evacuation, because the neck of the bladder and meatus would necessarily have been so stretched and injured, that I cannot help thinking the patient must have suffered immediate or remote injury.

The cutting operation which I performed was of the simplest nature, and, as generally happens (and this is a good practical point to bear in mind) in young children, the bladder wound healed directly, so that the child was well almost as soon as he would have been under the most favorable conditions, if the stone had been crushed. There was no danger of damage to his urethra, and we were certain of having got every bit of stone out of the bladder.

In the second case the reason was also clear. The stone was fixed in a cyst and was projecting only partly into the bladder. If I had attempted to crush the stone I should have crushed only that part which was projecting into the bladder, and should have left the remainder untouched. Some of the advocates of lithotripsy will tell you that there is not necessarily any excuse for cutting a patient merely because a stone is encysted; as it is said that with a little trouble, if the operator possesses the necessary manipulative dexterity, a stone may be extruded from the cyst in which it lies and subsequently be crushed. Theoretically this sounds attractive, and in a few cases may be practicable, but I am certain that in this case, at all events, I could not have squeezed the stone out of the cyst into the bladder without doing so much damage that it would have been a quite unjustifiable procedure. There is another reason why in cases of this kind lithotripsy is not good, viz. it is almost impossible to be sure of avoiding the chance of recurrence. The question of recurrence after lithotripsy is very important, and it arises particularly in connection with cases like that of the man with the stone encysted behind his prostate, and the young man who had the wandering stone. We have I think never yet got from the advanced lithotritists the proportion of recurrences of stone after crushing operations. For this there are without doubt very good reasons. Recurrence after lithotripsy of course, as a rule, does not take place until rather a long time after the operation,

and it is extremely difficult to keep patients under observation; moreover, it is particularly difficult to keep under observation patients who are disappointed with their treatment. The result is that some patients who, after having been crushed for stone, have had recurrent symptoms, apply to other people for relief. The patients argue, rationally enough from a certain point of view, that if the person who has performed the operation has failed to effect a cure, it is well to apply elsewhere on the chance of obtaining more success. This is not, of course, the case with all patients, but it certainly is with some. Therefore you may take it as being true that a certain proportion of the recurring cases do not return to the operators by whom their treatment was conducted. That recurrences do take place after these crushing operations there is no doubt, and it is in cases like two of those I have mentioned that recurrences are prone to happen. If I had been able to extract the stone from the cyst in the second or third case and had afterwards performed lithotritry, it must have been extremely difficult, with such a dependent pouch, however dexterous one may be, to be quite sure of preventing the gravitation of débris into the pouch. It is difficult to wash out these pouches effectually. You must bear in mind that, however small a piece of débris left behind may be, it is sufficient to form a nucleus of another stone.

I will now give you an instance of recurrence after a crushing operation by an expert lithotritist; it is not the only one I have met with, but it is sufficient to illustrate my meaning. A young man, 25 years of age, had a vesical calculus. It was crushed by a very skilful lithotritist, and all went well until about two months after the operation, when the patient began to have a return of some of his old symptoms. Thereupon he went back to the surgeon who had performed the original operation, and told him he thought there was a recurrence. But the operator declined to allow the possibility of such a thing. So sure, indeed, was he that there was no stone in this man's bladder that he would not even sound him. He was asked to do so in order to make sure of the state of affairs, but he said it was unnecessary. Well, the patient came away, followed the treatment suggested, and derived comfort for a short time. Then the symptoms began to increase, and I saw him about four months after the original

crushing. Upon passing the sound there was no question about the recurrence; there was a stone quite fixed at the left side of the base of the bladder. I could not move it at all. There was clearly only one thing to do to get rid of this stone. Crushing had failed before, and I felt certain would fail again. I therefore did a supra-pubic lithotomy, and found the stone firmly encysted; it was not larger than a couple of horse beans, and the nucleus was a fragment from the stone, which had no doubt dropped into the cyst at the original crushing. That is a good instance of recurrence after lithotritry. If that man had been cut in the first instance the stone would have been taken out whole, and no recurrence would have ensued. Since I removed the second stone the patient has had no symptoms, and as far as I know at present he is perfectly well, the operation I performed having been done four or five years ago.

The next point which arises in connection with this subject, assuming that cutting operations are frequently justifiable, and in my mind there is no doubt that they are—is, which is the best operation to use? Is the supra-pubic or lateral lithotomy the better procedure? For my own part I should not have thought the question worth discussing, but it so happens that from my experience of examinations I find that the majority of men who come up for examination if asked which operation is the safer, say lateral lithotomy; and some examiners I find hold the same view. They say that the supra-pubic operation is more serious than the lateral because in the former there is danger of wounding the peritoneum. That is the main reason I have heard given. I take it that the men who present themselves for examination would hardly give this reason unless they had been taught to regard the peritoneal danger as an important objection to the operation. Surely it is hardly rational to regard with so much fear the possibility of wounding the peritoneum in this particular operation. I am not one of those who think that even in these times we can deal with the peritoneum without any risk at all. But we all know that there is not necessarily a great deal of risk even if the peritoneum is wounded, if ordinary care be subsequently taken. But in this particular operation—supra-pubic lithotomy—why should the peritoneum be wounded? In most cases it is not

even seen during the operation. The greatest danger of wounding the peritoneum in supra-pubic lithotomy is caused by the use of metal retractors for holding out of the way the peritoneum and subperitoneal tissue. If, however, you use the best retractors of all, namely, two fingers of a reliable assistant, there is not the least danger of harm to the peritoneum. I have, of course, performed supra-pubic cystotomy a great number of times, but on very few occasions have I seen the peritoneum during the operation. Therefore, why this danger is regarded with so much apprehension I cannot think. The only other danger worth mentioning is the possibility of tearing the peritoneum during attempts to extract very large stones, but this danger is entirely obviated by crushing such stones before extracting them, which should, of course, always be done. The fear formerly entertained of profuse hæmorrhage from the parts about the anterior aspect of the bladder in this operation is quite groundless. I have never seen any hæmorrhage worth mentioning, and I do not think I have ever applied a ligature to any vessel about the region of the bladder. Occasionally, for the sake of cleanliness, it may be found desirable to put a ligature on a vessel or two in the abdominal parietes, but I have never had to ligature a vein about the bladder. A good deal of attention was at one time given to this question, and special instruments have been devised to enable the operator to avoid wounding the perivesical veins; a finger gently used is, however, always sufficient to push the veins aside. On the other hand, in the operation of lateral lithotomy there are dangers which the most expert lithotomists cannot avoid. And I have often told you before, the dangers of lateral lithotomy are out of sight, and sometimes quite unavoidable; a little abnormality of parts may lead to the wound of a vessel, which may bleed seriously, and compel one to adopt all sorts of measures which are not only disconcerting at the time, but delay the subsequent progress of the case. There is, in my opinion, little or nothing to be said in favour of lateral lithotomy. In the supra-pubic method the risks, such as they may be, are obvious and readily avoided, whilst the facility for completely clearing the bladder of foreign contents is complete.

The case of the female patient is rather an interesting one, the association of stone with

malignant disease of the bladder being rare. Supra-pubic cystotomy is not, perhaps, so common in women as other operations for extracting stone from the bladder. But even in females the supra-pubic method is, I think, the best cutting operation to perform, because it gives a good opportunity of examining the whole bladder and dealing with it properly. If I had chosen the crushing operation for this patient I might have detected the malignant disease, or I might not. If I had detected it I could not have dealt with it as I did after making the supra-pubic opening, because it happened that the disease was high up; and from the way in which the patient improved afterwards there is no doubt I must have done her a good deal of good by the thorough scraping away of the disease, which I could not have so well managed in any other operation.

Sometimes in dealing with stone in the bladder, whether by lithotripsy, supra-pubic cystotomy, or by lateral lithotomy, the difficulties are so great that the cases are practically incurable. A case of this kind which I had under my care for five or six years is perhaps almost unique. The patient was a man 47 years old, who first of all came into the hospital in 1890 with pronounced symptoms of stone with very acute pain. On sounding him his bladder seemed to be full of stones; there appeared to be any number of them. The pain was excruciating, and he had a good deal of cystitis. Crushing was out of the question. I cut him above the pubes, and took out eleven stones, varying in size from that of a chestnut downwards. He did very well; the bladder wound healed up very quickly, and he left the hospital at the end of ten weeks perfectly well. He came back in January, 1891, suffering from the same symptoms as before, and again I felt several stones in his bladder. Accordingly I cut through the cicatrix of the old wound and removed three more stones. In June, 1891, the same symptoms recurred, and I removed three stones again. To make a long story short, between January, 1891, and 1895 I removed from that man twenty-eight good-sized stones in five supra-pubic operations. He had still another recurrence, and he finally died of disorganisation of his kidneys. Whilst under treatment he had an attack of renal colic on the right side, which led me to think that the right kidney supplied the stones. After consulta-

tion with my colleagues, I suggested to the patient that it would be a good thing to cut down upon his right kidney, with a view to removing any stones it may have contained. He did not view the suggestion favourably, because he said he felt sure from his sensations that if he had stone in one kidney he had a similar condition in the other. The operation of exploring both his kidneys was, of course, a somewhat serious one, and he therefore declined any further operation. After his death we found both his kidneys were full of stones; I cannot say how many each contained. Doubtless the stones which were found did come from time to time from his kidneys into his bladder, giving rise to the recurrent symptoms, so that as far as the bladder symptoms were concerned he was incurable. Occasionally, therefore, a case of stone in the bladder may be met with in which recurrence is inevitable, no matter what treatment may have been employed. Under ordinary circumstances, however, there is no doubt that a certain cure is more likely to be effected by supra-pubic lithotomy than by any other method. Speaking generally, the supra-pubic operation has one great point in its favour, viz. it can be performed by any surgeon of ordinary capacity who understands the value of surgical cleanliness. Lithotripsy, on the other hand, will always be an operation which only the minority of surgeons can perform very skilfully. There is a certain amount of manual dexterity and aptitude required which a man cannot exactly learn. It is the same with the use of a catheter; some men, however good surgeons they may otherwise be, seem to be unable to pass a catheter properly, while other men seem to fall instinctively into the way of doing this without trouble; I suppose it is a question of aptitude. That being the case, lithotripsy is an operation which will be extensively performed with success by only a comparatively small proportion of surgeons. It follows, therefore, that an ordinary practitioner, whose experience can hardly be large, is likely to do far better for his patient and for his own reputation by adopting, excepting in the most simple and straightforward cases, supra-pubic lithotomy, than by performing, probably imperfectly, lithotripsy, which, although it may not be a cutting operation, is very apt to lead in comparatively inexperienced hands to damage of the bladder and tearing of the mucous membrane of the urethra by the passage of large

tubes, &c. One of the main objects I had in selecting this subject for discussion to-day was to enable me to express my views on the different methods for the treatment of stone in the bladder, and to state my opinion that for the ordinary surgical practitioner the best method at his disposal for the treatment of vesical calculus is, as a rule, supra-pubic lithotomy.

FEVER AND PROGNOSIS IN PHTHISIS.

BY

F. GRAHAM CROOKSHANK, M.D.Lond.

STRÜMPPELL in the course of March, 1894, differentiated five types of pyrexia which occur in chronic pulmonary tuberculosis, and has indicated the prognostic value of observation of the type in any particular case.

The five types, according to Strümpell, are—

- A. Morning temperature normal.
Evening temperature 100.4° — 101.3° F.
- B. Morning temperature normal or nearly so.
Evening temperature 101.3° — 104° F.
- C. Morning temperature 100.4° — 101.3° F.
Evening temperature up to 103.3° F.
- D. Continued fever.
- E. Fever of totally irregular type.

With type A the prognosis is relatively good; with B steady advance of disease occurs, though fair health may for a time be obtained; with C the prognosis is more unfavorable, lobar disease frequently being present; D is found chiefly in cases of acute onset, and the prognosis is bad. Type E characterises the whole duration of disease in some cases, but only the later stages of others. The prognosis is extremely bad.

Strümpell further states that in all cases of pyrexial phthisis fever invariably assumes one and only one of the above forms. Any change in type is due to intercurrent disease, or complications aggravating the tubercular process.

Through the kindness of Dr. Percy Kidd I have had the opportunity of examining a large number of the notes and temperature charts of his patients at the Brompton Hospital for Consumption, and of comparing the results with Strümpell's statements.

The result confirms Strümpell's statements in almost every particular, although exceptionally, especially in cases of the "emphysematous type of phthisis," an almost apyrexial temperature chart is found to accompany the steady progress of disease.

The commonest type of fever met with at the Brompton Hospital is Strümpell's type A, probably because those individuals are preferred for admission who are most likely to improve.

The limits of this type, however, are rather wide, and, if it be subdivided into two groups, in

otherwise in the different groups, and in the last column the average period of observation of each case is given. Of the cases not improving in class A (i and ii) twenty-two out of thirty-seven had irregular fever within the limits of their type, and the remainder were complicated by laryngeal or intestinal disease.

Although only a relatively small number of cases are here tabulated, a much larger number were observed, and gave practically the same result.

	Number of Cases.	Improved.	Did not improve.	Deaths.	Average Gain or Loss.	Period of Observation.
A i	72	59=81.9 per cent.	13=18.1 per cent.	1=1.3 per cent.	+3.7 lbs.	65.3 days.
ii	37	13=35.1 "	24=64.9 "	3=8.1 "	-.8 "	71.9 "
B	20	6=30 "	14=70 "	4=20 "	-3.1 "	71.8 "
C	8	8=100 "	3=37.5 "	73.9 "
D	7	7=100 "	6=85.8 "	25. "
E	6	6=100 "	6=100 "	50. "

one of which the daily range of temperature does not, and in the other does, exceed two degrees, nearly all the cases improving are found in the former subdivision.

In any particular group of cases of one type the prognosis appears to be favorable in ratio to the regularity of the temperature. For example, a case of type B with a regular temperature chart will probably do better than a case of which the temperature is irregular within the limits of type A, other things being equal.

Nearly every case in which the "typus inversus" of fever is noted falls within Strümpell's group C, and this accords with the fact that such cases have run generally a rapid course, and show disseminated disease of the lung.

In almost every case in which the type of fever changed under observation the variation in type coincided with the onset of complications, usually laryngeal or intestinal. Not uncommonly the change in type follows a severe hæmoptysis.

In the following table 150 cases (which were observed under approximately the same conditions) are grouped according to the type of fever present in each case, class A being subdivided as suggested above.

The average gain or loss in weight of each patient during the period of observation affords some means of comparing the improvement or

NOTES.

Disseminated Infectious Gangrene of the Skin in Children.—Médéric Caillaud ('Revue Mens. des Mal. d. l'Enfance,' Jan. and March, 1897).—This form of gangrene has some special features which distinguish it from other forms of gangrene, as diabetic, toxic or trophoneurotic.

It appears under two different conditions: First, there may be a previous ulcerative skin lesion, from and upon which the gangrene starts. Second, the gangrene may appear immediately after a non-ulcerative manifestation of the skin, and it is not an occasional accident, but it constitutes an essential element of the disease. All causes which will put the child in unfavourable conditions and impair its general health predispose to that contagious affection, and especially all sorts of existing ulcerations and erosions may serve as a door of entrance for the contagion.

There are reported cases of so-called primitive gangrene of the skin, when it was entirely free from erosions, but not in an absolute healthy condition, as in cases of erythema nodosum, with or without purpura.

Upon the nodules of erythema nodosum or upon purpuric plaques appear pin-head size vesicles filled with an opaque liquid. In twenty-four hours

the vesicle is changed into a pustule, surrounded by a dark brown border and a red, several cm. wide, outside zone. The next day the pustule reaches the red outside border. On the fourth a dark crust, adherent to the deep tissues, is formed, surrounded by a greyish border, which separated the necrotic from the inflamed tissues. The greyish border is surrounded by a red zone, and the skin around the zone is inflamed, swollen, and gives to the touch a feeling of skin of a corpse preserved in ice.

In eight to fifteen days the crust falls off, and a deep circular ulceration is visible. The sore is always circular, unless it follows an application of mustard plaster, when it is serpiginous. In forty days a scar is formed.

It is mostly localised upon the lower parts of the abdomen, upper parts of the buttocks. Sometimes the face and scalp are affected. The hands and feet are generally left free.

It is accompanied with severe general symptoms, fever, vomiting, and with albumen in the urine. With the appearance of the gangrene, the fever disappears. Developing upon existing skin lesions as varicella, vaccine, impetigo, it follows mostly the same course, with slight variations. In most of the cases, especially when long complications intervene, the prognosis is unfavorable. Without complications, the prognosis is more favorable if the child is advanced in age. Iritis, kerato-conjunctivitis, and purulent arthritis are also common complications. Bacteriological researches show that the disseminated gangrene of the skin is due to a pyogenic infection of the skin, helped either by one particular microbe, which has not been isolated up to the present time, or by a thrombus of the vesicles, which impaired the nutrition of the skin. The treatment consists of daily baths in a 3 per cent. solution of boracic acid, in 1:10,000 solution of sublimate of mercury, followed by an application of boracic acid solution or carbolic water. When the crusts are off, antiseptic powders are indicated. Iodoform-collodium is to be avoided.—*Pediatrics*, July, 1897.

Acute Oedema of the Larynx.—Stankowski relates two cases due to iodide of potassium. 1. A man æt. 28 years was suffering from pulmonary and laryngeal phthisis. The laryngeal manifestations consisted in infiltration of the interarytænoid

and right aryæno-epiglottic folds. He was treated with potassic iodide with the view of improving the nutrition. In five days a considerable oedema of the left aryæno-epiglottic fold was noticed, which disappeared when the iodide was discontinued. The author gives his reasons for believing that this oedema was due to the iodide, and was not a manifestation of tuberculosis. 2. This case occurred in a man æt. 31 years, who had been treated with several courses of mercurial inunction. Some six years after the initial lesion he was given potassic iodide, as syphilitic manifestations had again appeared, as well as a solution of iodide in potassic iodide to be applied to a swelling on the lip. After he had been taking the iodide for two weeks he began to suffer from coryza and dyspnœa. There was an oedematous swelling on the right side of the larynx. The iodide was omitted, and the swelling in the larynx soon disappeared. In this case there was no infiltration, and the swelling had not the characteristics of a syphilitic perichondritis. The oedema was unilateral in both cases,—a feature not often met with in cases hitherto recorded. In the first case 12 grammes (3 drachms) of iodide were taken in five days; and in the second case iodide had previously been taken without any evidence of iodism. In both cases the oedema did not appear at the commencement of the treatment with small doses of iodide, as has generally been known to be the case. The laryngeal symptoms were never urgent, as has happened in most of the recorded cases.—*Münch. med. Woch.*

Test of the Permeability of the Kidneys.—Methylene blue injected subcutaneously after urinating appears in the urine normally in half an hour, attaining its maximum of intensity in three to four hours. But if the kidneys are affected the blue appears much more slowly, and in seventy-seven observations this test confirmed in every case the diagnosis of renal disturbances. Achard and Castaigne also found several cases in which there were no clinical evidences of renal trouble, but investigation in each case disclosed early troubles. They consider the test a valuable confirmation of other evidence, as the function of the organ is alone involved. The average dose injected was 0.050 gramme.

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A CLINICAL LECTURE

ON

ENLARGEMENT OF THE HEART

Delivered at the Brompton Hospital by

J. MITCHELL BRUCE, M.D., F.R.C.P.,

Consulting Physician to the Hospital.

GENTLEMEN,—Enlargement of the heart is a subject which necessarily commands the interest of the practitioner for its own sake, but it is of particular interest indirectly because in every instance, after its existence has been discovered, a further diagnosis must be made, namely, of the cause of the enlargement. Enlargement of the heart is a secondary condition, and it ought always to make us search for a primary lesion. Again, in addition to the existence and origin of cardiac enlargement, there are the different forms or kinds of enlargement to be differentiated, and their respective clinical significance understood. Hypertrophy has one significance, hypertrophy with dilatation has another significance, and dilatation in cardiac failure has an entirely different significance again from the other two kinds. Not until the existence, origin, and kind of enlargement have been appreciated, are we qualified to apply the principles which ought to guide us in the prognosis and treatment of diseases of the heart, the circulation, and the kidneys. Nowadays everybody is well practised in the examination of the heart, but I doubt whether enlargement of the heart is always carefully studied in ordinary practice. Many observers are content with arriving at the diagnosis of a murmur. There is a certain amount of trouble attached to accurately mapping out the size of the heart, but so great is the importance of the observation that one cannot insist too strongly upon making it a matter of clinical routine.

This afternoon I propose to show you a few cases of enlargement of the heart, and in so doing to demonstrate some of the morbid conditions with which it is associated, and to discuss very

briefly the significance of the different forms and certain indications which they furnish for treatment.

First, let us examine this patient, whom I show you as an instance of simple hypertrophy of the left ventricle. The primary disease is chronic nephritis, but as there is also emphysema this is not quite so typical a case of simple hypertrophy of the left ventricle as we might have wished it. You observe that I first determine whether there is any precordial bulging, the extent and other features of the precordial impulse, the exact situation and character of the apex-beat, the transverse and vertical precordial dulness; and finally I auscultate. The conclusion at which I arrive is that the left ventricle is moderately large and powerful, and that the valves are sound.

In my next patient, instead of enlargement of the left ventricle there is enlargement of the right ventricle. This lad is the subject of pure mitral stenosis. He presents a different precordial dulness from the one we have just been studying; here there is a well-marked dull area corresponding to the situation of the right ventricle. Theoretically we consider there is pure hypertrophy of the right ventricle; as a matter of fact, there may be some dilatation as well.

The third case is one of aortic incompetence. The enlargement of the heart no longer consists in simple hypertrophy of the left or of the right ventricle, but in dilatation with efficient hypertrophy of the left ventricle. Looking at the man's chest you notice at once marked precordial bulging, and we gather from his history that at the age of four he had rheumatic fever. Note also the impulse, the character of the apex-beat, displaced far to the left and downwards, the different area of dulness from before, and the characteristic murmur. This is not simple hypertrophy; it is dilatation with hypertrophy—a hypertrophy which is efficient, for the man presents no evidences of distress.

In the next case, you again discover that there is aortic incompetence as in the last instance, with well-marked signs of cardiac enlargement. But

note, the patient is in bed and in considerable distress. The patient has the characteristic "cardiac" aspect. A flush is seen upon the anæmic face, a yellow tint suggests hepatic congestion, and the expression is one of anxiety. Therewith we have precordial distress or even pain, orthopnoea and other disturbances of respiration, cough, expectoration, and perhaps actual hæmorrhage from the lungs. It is a perfectly different clinical picture from that seen in the other two conditions, and the signs are different and distinctive. There are the evidences of enlargement, but instead of the thrusting impulse of pure hypertrophy, instead of the heaving impulse of dilatation with compensatory hypertrophy, there is, in this third class, hardly any impulse at all; the veins are distended; the blood is dammed up in the liver; the extremities and serous cavities are dropsical; and I may add that the urine affords abundant evidence of renal congestion.

I want you to notice very particularly the difference between the three conditions as illustrated in these patients. The man with primary Bright's disease and simple hypertrophy has come here, has shown himself and has walked about; so also has the lad with mitral stenosis, and so has the first patient with aortic incompetence and great dilatation with efficient hypertrophy. But when we turn to the case which I have had to show you in bed, we are brought into relation with the third form of cardiac enlargement, a condition which is incompatible with getting about in comfort or even safety. The patient I show you in bed proves to be the subject of cardiac dropsy. You notice the swollen legs and the enlarged liver; there is some effusion into the peritoneal and the right pleural cavity. This is very different from the other patients. As you have seen for yourselves, they appear to have very little to complain about. They are obviously the subjects of vascular and cardiac disease, with compensation or something approaching it. So long as they keep quiet they are fairly well. But this patient in bed has lost his compensation. Here, then, we have dilatation, but it is dilatation with failure of the heart. The hypertrophy is no longer efficient. We have discovered a third kind of enlargement of the heart. It is not simple hypertrophy; it is not dilatation with efficient hypertrophy: it is dilatation with an inefficient heart.

Different names might be proposed for this kind of enlargement of the heart, but the name most generally used is "dilatation with failure," or simply "cardiac failure."

There are, then, these three kinds of enlargement of the heart; first, *simple hypertrophy*, such as you find in chronic Bright's disease and in pure aortic obstruction; second, *dilatation with efficient hypertrophy* and complete compensation; and third, *dilatation with failure* and cardiac dropsy.

You will now ask me, "What is the good of knowing and distinguishing these three kinds?" This is the very point of my lecture, and may now be confidently approached. Let us consider for a moment the doctrine of cardiac enlargement. What does it signify? Before we can grasp this, I must ask you to let me remind you of a few points in the dynamics of the circulation. You know we have three principal forces at work in connection with the circulation—we will speak only of the left ventricle. First there is the driving power of the heart, which is exerted by the myocardium during systole. Secondly, we have the weight or load of blood within the heart. This blood may be called the "charge" of the ventricle, just as we speak of the charge of a gun. The third force is the resistance to the discharge. There is some resistance within the heart, but the chief resistance is in the arteries, beginning at the aortic valves, which are weighed down by a certain pressure—the blood-pressure. The business of the left ventricle is to exert such force in the systole as shall drive this charge of, say, six ounces of blood, right into the mouth of the aorta, and effect its complete circulation. This is the primary notion. The next point to appreciate is that if more work be required of the ventricle, in consequence of some increase in the charge of blood, or of increased resistance in the organs of circulation, an increase of force is displayed in exact relation to the demand; and if this continue, *hypertrophy* is the result. If there be any condition forcing the heart to hold more blood than normal, then increased force is evoked, and after a time hypertrophy results; that is to say, hypertrophy accompanies dilatation from overfilling. On the other hand, if the dynamic disturbance consist in increased resistance at the aortic valves, hypertrophy pure and simple is developed, as we

see in chronic Bright's disease and in aortic stenosis.

Now we pass on to *dilatation*, and I will ask you to consider in how many ways the left ventricle can become dilated. How comes it to have more than the normal amount of blood accommodated within it? In three ways only. The first way is when the chamber is filled from two sources at once—from the left auricle normally, and from the aorta abnormally, through aortic incompetence. The heart has to hold more; it is stretched; it accommodates itself to hold; and then it propels a bigger bulk and greater weight of blood. With this heavier weight to drive hypertrophy comes on; and thus along with dilatation from double charging you have hypertrophy—dilatation together with hypertrophy—and compensation.

The second way of overfilling in which the left ventricle becomes dilated, is by receiving an excessive quantity of blood through the mitral orifice. This happens, of course, in mitral regurgitation, the commonest of all our clinical types of cardiac trouble.

But there is a third way, and it is very important, in which the left ventricle may become over-distended and thus dilated. If in anticipation of the charge it will receive from the left auricle it have already within it a residuum of undischarged blood from the previous systole, that is, if the previous systole have been not so completed as to sufficiently empty the left ventricle, but have left an undischarged amount of blood within it, then the charge of blood from the left auricle is added to the arrears, and the chamber is dilated. The dilatation does not originate in overfilling; it originates in *incomplete emptying*. It is the outcome of cardiac failure. It is enlargement of the heart, due not to overfilling, but to under-emptying. In the first two ways you have a purely mechanical dilatation, and therewith a compensatory hypertrophy. But this has a physiological origin: it is due to functional weakness of the cardiac wall itself.

I trust you understand this point, because on it are founded the principles of the prognosis and treatment of valvular disease and of failing compensation in chronic Bright's disease. The patient with hypertrophy will continue comparatively well for an indefinite time, that is, as long as you maintain the hypertrophy and the primary disease

does not increase. In cardiac dilatation with efficient hypertrophy, the second form, you have a more precarious condition; but provided the conditions of hypertrophy are maintained (good blood, good food, and not too much work), compensation may here also go on indefinitely. It is a wasteful process; still, it is the best that can be done. But when we come to the third condition, and the prognosis and treatment of cardiac failure, we start with the myocardium broken down. Of course, of itself, this is as bad as can be. It is ruin for the patient unless you come to his assistance. What can you do? It is here that art steps in. In the first place you must undo, if possible, the *cause* of the failure of the heart. You will put a stop to muscular overwork. You will restore the blood. You will interdict poisoning of the heart's wall with tea and tobacco, and eliminate intrinsic toxines and uric acid. You may succeed in relieving the heart from nervous strain. Having thus or otherwise attended to the causes of the breakdown, whilst doing so, you proceed to act on the tissues of the heart with digitalis and strychnine, and thus get more force out of the ventricle. Still more quickly you could reduce the size of the ventricular load by bleeding, or you could purge your patient and relieve that long train of backward congestion and dropsy. By fulfilling these different indications, you can, as you know, quite often rescue your patient even from the desperate condition of dilatation with failure.

CEREBRAL LOCALISATION.

BY

W. H. B. STODDART, M.B., B.S.,

Late Resident Medical Officer of the National Hospital for the Paralysed and Epileptic, Queen Square, Bloomsbury; Clinical Assistant at Bethlem Royal Hospital.

IN the following paper I have endeavoured to set down as briefly as possible the chief points which guide us in the localisation of an intra-cranial lesion, taking up those points *seriatim* as they arise in the systematic examination of a patient supposed to be suffering from coarse brain disease.

In no branch of medicine is a knowledge of anatomy more necessary than here; and that person is most likely to localise a lesion correctly who is most intimate with the anatomy and physiology of the brain, when the problem of a case presents itself clinically.

Taking *mental symptoms* first—These have practically very little localising value; they may arise in connection with a growth anywhere within the cranial cavity, and this is not to be wondered at, because they arise even with diseases of the heart, lungs, and stomach. Much more, then, should we expect to find them in disease of central nervous system.

Roughly, however, one may say that disease of the frontal lobes causes loss of memory and moral deterioration; while disease of the posterior third of the hemispheres is liable to cause mental symptoms not unlike those of a general paralytic.

Let me here say, too, that hallucinations of vision are of little localising value. Hallucinations of the other senses may possibly be more important.

Persistent *headache* should lead you to think of some intra-cranial lesion, but the situation of that headache is of little localising value. Intense frontal headache may be due to a cerebellar tumour, and it is generally on the opposite side in such cases. If the patient complain of headache (so-called) at the nape of the neck, you will probably be right if you conclude that the tumour is subtentorial, or at least not higher up or farther forward than the optic thalamus.

Cephalic tenderness may be some help. If the tenderness be deep (brought out only on percussion or firm pressure), the tumour is in the membranes somewhere near the tender spot, and, *cæteris paribus*, probably operable. If the tenderness is superficial, the tumour is in the brain substance, cerebral tumours causing tenderness of the scalp, cerebellar tumours causing tenderness of the face.

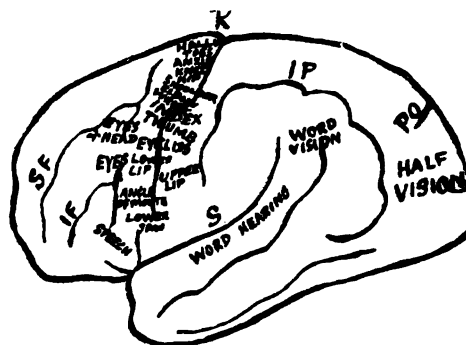
If the patient complain of *giddiness* one naturally first thinks of the cerebellum; but I only mention this symptom to warn you against attaching too much localising value to it.

Insomnia, delirium, stupor, and coma alike have no localising value.

I now come to the consideration of *fits*,—not ordinary epileptic fits, for they are valueless for localising purposes. The presence of worms in

the rectum is almost as likely to cause them as a tumour of the frontal lobes. Matters are different with epileptiform or Jacksonian fits. These are frequently of the utmost value in determining the situation of a cerebral tumour. What you especially want to ascertain is how the fit begins. An ordinary epileptic fit begins usually with loss of consciousness; an epileptiform fit never begins with loss of consciousness, and hence the patient is usually able to tell how his fit begins. And this is important, for a fit does not always start with an outward and visible sign, but often with a local subjective sensation more or less complex; for instance (*a*) seeing complicated pictures, such as the classical "little old woman in a red cloak picking up sticks," (*b*) hearing church music or the ringing of bells, (*c*) a metallic taste, or (*d*) a smell of something burning, or (*e*) a sensation of numbness starting, for example, in one toe. In such cases you would localise your lesion in (*a*) the angular gyrus, or (*b*) the superior temporo-sphenoidal convolution, or (*c*) the hippocampus major, or (*d*) the uncinate gyrus, or (*e*) the upper end of the fissure of Rolando.

If the onset is with movement, you then only require a mental picture of the following diagram before you, and your localisation is complete except for the depth of the lesion. To this I will refer later.



S F.—Superior frontal sulcus.
I F.—Inferior frontal sulcus.
R.—Fissure of Rolando.
S.—Sylvian fissure.
I P.—Intraparietal sulcus.
P O.—Parieto-occipital fissure.

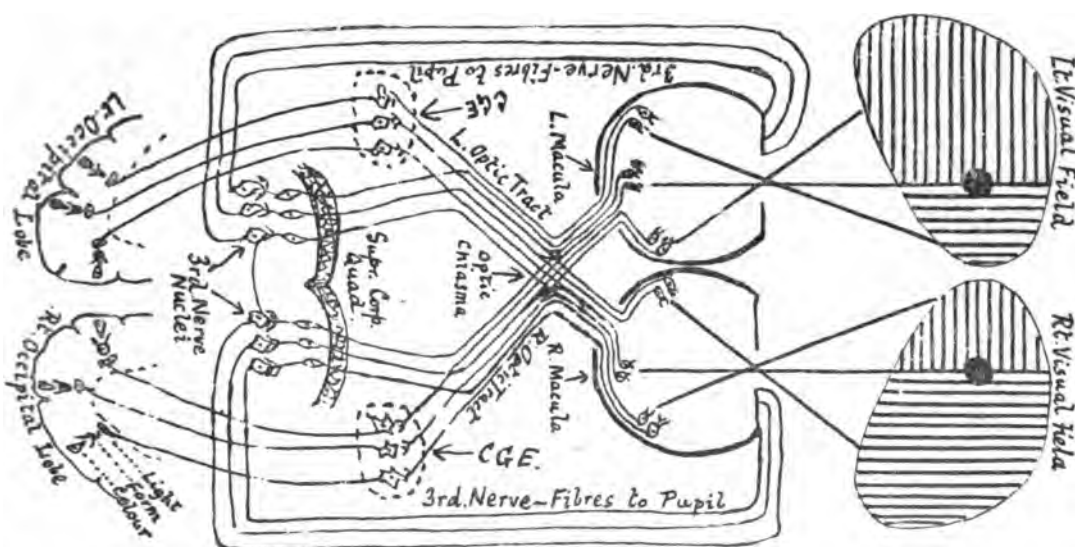
A few points about the cranial nerves :

(1) Loss of the sense of smell is usually due to pressure on the olfactory tracts, due in turn to presence of a tumour in the frontal lobes; but it

is also said to have been lost in some cases of cerebellar disease. If the "anosmia," as it is called, is due to pressure on the tracts, as it usually is, the lesion is upon the same side of the brain. The symptom is, of course, valueless if there is disease of the nose itself, but it is also valueless in affections of the fifth nerve, because the Schneiderian membrane is not then kept in its normal supple and moist condition. The nose should be systematically sprayed out with glycerine and water before testing the sense of smell in a case of this kind.

(2) Optic neuritis is a moderately constant sign of intra-cranial tumour, but its localising value is small. The most intense optic neuritis occurs

condition in which a patient is blind to one half of the visual field, and should always be looked for in a case of intra-cranial disease,—especially if the patient complain of blindness of one eye. When hemianopia occurs, it is usually absolute; in other words, the patient can see *nothing* in the hemianopic field: but hemianopia may be, so to speak, specialised for colour only or for form. This is *supposed* to be due to affection of different layers of the cortex. Right hemianopia (*i. e.* blindness of the right half of the visual field), is, of course, referable to the left half of each retina (left occipital lobe), and *vice versa*. And let me say that the blindness does not always come up to the middle line.



where the tumour is in the cerebellum or occipital lobes. Tumours at the base of the brain are not so likely to cause optic neuritis, but they sometimes do.

Lesions of the angular gyrus have been said to cause crossed amblyopia, but this idea is now abandoned.

Complete blindness due to intra-cranial mischief is (apart from optic neuritis and atrophy) most likely to be due to a lesion in one of two places, either the optic chiasma or the splenium of the corpus callosum, where the optic radiations from both occipital lobes would be caught.

Hemianopia is due to a lesion somewhere between the optic chiasma and the occipital lobe of one side, or in either of these two places. It is that

The best explanation of the fact that the macula escapes as a rule is one based on the supposition that each macula receives a supply from each occipital lobe. And the fact that in some cases the limit line passes through the macula is explained on the hypothesis of some individual variation in this respect.

Working from behind forwards—a large lesion of one cuneus will cause blindness of the opposite half of the visual field. The distinction between hemianopia due to a lesion in the occipital cortex, and hemianopia due to a lesion of the optic radiations at the posterior part of the internal capsule, depends on the proximity of the latter fibres to the sensory tract. In other words, hemianopia due to a lesion of the optic radiations is associated

with hemianæsthesia, while that due to a lesion of the occipital lobe is not. Lesions of the superior corpora quadrigemina do not cause hemianopia. Those bodies subserve the function of movements of the eyes, and not of vision.

A lesion of the external geniculate body (C G E), on the other hand, does cause hemianopia. Here again we have hemianæsthesia and also some hemiplegia, but from pressure on the crus. The diagnostic sign, however, is involvement of the oculo-motor nerves. To repeat, a tumour near the external geniculate body may be diagnosed if there is hemianopia associated with ophthalmoplegia of the opposite side.

The diagnostic sign of a lesion of the optic tract is the hemianopic pupil. This sign is present when the pupil does not contract to light thrown on the blind half of the retina from an ophthalmoscopic mirror.

Lesions of the chiasma cause irregular forms of hemianopia (bi-temporal, altitudinal, &c.)

(3) A lesion of the third nerve causes paralysis of the levator palpebræ and of the internal, superior and inferior recti as well as of the inferior oblique. All this is called external ophthalmoplegia. It also causes internal ophthalmoplegia or paralysis of the sphincter iridis, and of the ciliary muscle.

To put all this clinically—a lesion of the third nerve causes paralysis of the accommodation, so that the patient cannot see near objects distinctly or read small print. The pupil is dilated and does not contract to light, or with an attempt at accommodation or convergence. There is divergent strabismus, and the patient cannot turn the eye upward, downward, or inward. Finally, there is ptosis. If the lid be raised the patient has diplopia, crossed so that the right image is seen with the left, and the left with the right eye. Involvement of the third nerve must obviously be associated with a lesion at the base of the brain.

The diagnosis between a lesion of the trunk and one of the nucleus of the third nerve depends upon the fact that the inner or palpebral fibres of the orbicularis palpebrarum are supplied by nerve fibres which arise in the nucleus of the third, but pass down along the posterior longitudinal bundle, and emerge among the fibres of the facial nerve. Hence a lesion of the nucleus of the third nerve causes weakness of the power of closing the eyes naturally. The patient may be able to screw up

his eyelids tightly, but if told to close them naturally he can offer no resistance to an attempt made to raise the eyelid with the finger.

(4) With regard to the fourth nerve I will merely mention that the diplopia is homonymous.

(6) The sixth nerve has such a long course within the cranium that paralysis of the external rectus (causing convergent strabismus) is not regarded as a very reliable sign for localising purposes unless other signs point to damage of the sixth nucleus. The diagnosis of lesions of the sixth nucleus depends upon the following considerations.

Normally the internal rectus of one eye acts in concert with the external rectus of the opposite eye on looking to one side. Hence, as might be expected, a connection exists in a tract of fibres, which passes along the posterior longitudinal bundle from the sixth nucleus to the third nerve of the opposite side. And hence it follows that a lesion of the sixth nucleus in the pons causes not only weakness of the external rectus of the same side, but also weakness of the internal rectus of the opposite side. That is to say, that there is weakness of the power of looking to the same side. A lesion of the trunk of the sixth nerve does not do this. If both sixth nuclei are involved,—as they often are, being so close together,—the patient is able to look upwards and downwards, but not to either side.

(5) Lesions of the fifth nerve cause, on the motor side, paresis of the muscles of mastication (temporal, masseter, pterygoids and suprahyoid muscles) and, on the sensory side, loss of sensation of the face and scalp in front of a coronal line joining the two ears. They cause anæsthesia of the nasal cavities (except round the Eustachian tubes), and of the mouth as far back as the tonsils and soft palate, and they cause loss of taste for the front of the tongue. If you tell a patient so affected to open the mouth widely, the jaw deviates to the paralysed side. The nerve is usually caught near its nucleus at the upper part of the pons.

(7) A lesion of the trunk of the facial causes inability to raise the forehead on the same side, inability to frown on that side, or to close the eye or raise the upper lip. It is not usually caused by intra-cranial disease, but by cold or middle ear disease.

Paralysis of the face due to a cerebral lesion is

limited to the lower part of the face, and is usually only noticeable in an inequality of the naso-labial folds when a patient attempts to show his tongue or his teeth. The frontales are unaffected, and also the power of closing the eyes in the usual way, this movement being supplied from both hemispheres. But if you ask the patient to wink one eye, that is a specialised unilateral movement, and he is unable to do so on the paralysed side.

I have already referred to one set of fibres which comes by way of the facial nerve without arising in the facial nucleus, viz. those fibres which arise from the third nucleus to supply the inner fibres of the orbicularis palpebrarum. I now refer to another set of fibres analogously situated, which arise from the hypoglossal nucleus, pass up the medulla, and leave it by way of the facial nerve to supply the orbicularis oris muscle.

From this arrangement it follows that the innermost fibres of the orbicularis oris muscle are paralysed in affections of the hypoglossal nucleus. Such a patient is unable to whistle. And conversely, if a lesion is limited to the cells of the facial nucleus, the inner fibres of the orbicularis palpebrarum and oris will not be paralysed. Such a patient would be able to whistle, and to close his eyes naturally.

If doubt still remain as to whether the lesion is above the nucleus or not, the point is settled absolutely by the electrical reactions. These are unaltered if the lesion be supra-nuclear, but altered in the direction of the reaction of degeneration if the lesion be nuclear or infra-nuclear (nerve trunk).

(8) With regard to defects of hearing, I need scarcely say that they can only be depended upon for localising purposes when the conducting apparatus of the ear is intact.

The question as to whether the lesion is in the auditory centre or auditory nucleus or nerve is to be decided by other associated conditions.

(9) The glosso-pharyngeal nerve supplies the pharynx and back of the tongue with sensation. It essentially subserves the reflex of deglutition, so that if both ninth nerves are involved the patient requires feeding with a tube.

(12) The tongue may be paralysed on one side from involvement either of the tongue centre or of the hypoglossal nucleus or nerve. In any case, when protruded it deviates to the paralysed side.

If the palsy be cerebral there is no wasting or

change in electrical reactions as there is in the other two cases.

I have already referred to one point on which the different diagnosis rests between lesions of the nucleus and those of the trunk of the hypoglossal. In nuclear palsies the patient is unable to whistle on account of paralysis of the inner fibres of the orbicularis oris, which are supplied from the hypoglossal nucleus through the facial nerve.

In intra-cranial disease paralysis of the limbs usually takes the form of hemiplegia. In lesions of the pons there may be paralysis of all four limbs.

Anæsthesia for the most part coincides with the hemiplegia. Absolute hemianæsthesia almost certainly means involvement of the posterior end of the internal capsule, and the diagnosis is absolutely certain if this is associated with hemianopia.

As regards the *knee-jerks*, the following is not a bad clinical rule:—that the knee-jerk is diminished or lost in irritative lesions of the cerebrum, and paralytic lesions of the cerebellum, and it is increased in paralytic lesions of the cerebrum and irritative lesions of the cerebellum. This is by no means constant, nor does it entirely agree with experimental evidence. All that is claimed is that it may be taken as a useful clinical rule.

Aphasia is too vast a subject to deal with here. I will only say that it points to affection of the left cerebral hemisphere in a right-handed man, and to affection of the right cerebral hemisphere in a left-handed man. And by aphasia I mean inability to make or to understand written or spoken propositions; I do not refer to mechanical difficulty of articulation due to disease of the lower medullary centres, or to difficulty of writing due to paralysis of the hand.

Double hemiplegia is also a subject into which one cannot enter here. It differs from an imaginary condition, in which the patient is suffering from hemiplegia on both sides, in that the bilaterally acting muscles are affected. For instance, such a patient may be unable to swallow, although there is no lesion lower down than the cerebral hemispheres.

Anæsthesia due to disease of the cortex is but slight. The part that appears clinically to subserve this function is the cortex of the parietal lobe immediately behind the motor area. Lesions in this situation also give rise to loss of the sense

of position of the part represented there. And let me here draw attention to the difference between cortical and subcortical lesions. Tumours of the cortex are liable to give rise to Jacksonian fits; so are tumours immediately beneath the cortex, and so are tumours of the membranes pressing upon the cortex. Now in tumours in this part of the cortex, loss of the sense of position is a marked symptom. In the other two instances it is only a marked symptom for about a quarter of an hour after a fit; so that when you are called to a case of Jacksonian epilepsy, the first thing to be done, if the patient has just had a fit, is to test the sense of position of that part which was first affected during the fit. If the sense of position improves after an hour or so, the lesion is either subcortical or supracortical; if not, it is cortical. Local tenderness of the scalp is the next thing to be looked for in deciding if the lesion is in the membranes, and therefore an easy condition for operation (*vide supra*).

Lesions of the *corpus callosum* cause no special symptoms of their own. These lesions are diagnosed from consideration of symptoms due to involvement of the neighbouring structures such as the internal capsule.

A lesion of the corpus callosum is to be suspected in cases where unilateral fits are associated with hemiplegia of the opposite side.

There is some evidence in support of the view that the *lenticular nucleus* has something to do with the regulation of body temperature, and lesions here may *per se* cause a rise in temperature. The hyperpyrexia due to a hæmorrhage into the pons is a well-known phenomenon.

Mobile spasm of one side associated with hemiplegia of the opposite side is a combination which should lead one to think of the *optic thalamus*.

Ophthalmoplegia and a staggering gait with increased knee-jerks form the combination for the *corpora quadrigemina*. The ophthalmoplegia is due to involvement of the oculo-motor nuclei, and it is now generally accepted that the staggering gait is not due to backward pressure against the cerebellum, but to disease of the *corpora quadrigemina* themselves. Rotatory nystagmus sometimes occurs.

Crossed or alternate hemiplegia is a term applied to that condition in which there is paralysis of one or more cranial nerves associated with hemiplegia of the opposite side. Lesions giving rise to this

condition are, as a rule, easily localised. For instance, anosmia (loss of smell) or blindness (not due to local conditions in the nose or eye) of one side associated with hemiplegia of the opposite side point to tumours in the anterior part of the cerebral hemisphere pressing downward on the first or second nerves.

Third nerve paralysis of one side with hemiplegia of the other side means a lesion of the *crus cerebri*.

Paralysis of the fifth, sixth, seventh, or eighth nerves of one side with hemiplegia of the other side means a lesion of the pons.

Paralysis of the ninth, tenth, eleventh, and twelfth cranial nerves of one side associated with hemiplegia of the other side means a lesion of the medulla oblongata.

A word in conclusion about diseases of the *cerebellum*. Inco-ordination ranks first as a symptom of cerebellar disease. A lesion of the anterior part of the middle lobe causes the patient to have a tendency to fall forward, while one in the posterior part gives a tendency to fall backward. Disease of the middle lobe, further, gives rise to nystagmus, which may have some unusual character—for instance, it may be rotatory or oblique. If it is lateral and more marked to one side than the other, it is a guide as to which side of the cerebellum is affected. The nystagmus is more marked from the opposite toward the side of the lesion; e.g. in disease of the right side of the middle lobe, the nystagmus will be more marked to the right. Further, there is weakness of the back muscles in disease of the middle lobe.

If the lateral lobe is affected, there is a tendency to rotate towards the side of the lesion, so that the patient's face will tend to turn to the right in a case of disease of the right lateral lobe of the cerebellum. If the lesion involves the superior peduncle chiefly, this rotation is most marked in the head and neck; if the middle peduncle, the rotation is more marked in the trunk.

The position characteristic of disease of the inferior peduncle is that with rigid extension of the limbs of the same side with flexion of those on the opposite side.

There is frequently weakness of the limbs on the same side as the lesion, and also weakness of the power of turning the eyes to that side.

The knee-jerk is usually diminished on the side

of the lesion. Less commonly it is increased, in accord with the observations in experimental investigations on animals.

The chief difficulty in diagnosis of diseases of the cerebellum lies in the fact that you may have almost exactly the same clinical picture from disease of the opposite frontal lobe.

A CLINICAL LECTURE

ON

CANCER OF THE BREAST.

Delivered at the Bristol General Hospital, May 26th, 1897.

BY

CHARLES A. MORTON, F.R.C.S.,

Surgeon to the Hospital and Professor of Surgery in University College, Bristol.

WE have lately had four cases of cancer of the breast in Guthrie Ward at the same time, and as three of them present points of special interest, I propose to consider them as a sequel to a clinical lecture last winter on the differential diagnosis of swellings in the breast. In that lecture I described the diagnostic characters of scirrhus of the breast, and I wish now to draw attention to some unusual features present in these cases.

One case is remarkable as an instance of intense œdema of the breast, and redness of the overlying skin, associated with cancer in the breast. When the patient presented herself in the out-patient room, and I looked at her breast, I thought she had a very acute abscess, but as soon as I placed my hand on it and discovered the stony hardness of the swelling which lay beneath the red skin, and the firm mass in the axilla, I came to the conclusion we had one of those rare cases of malignant disease of the breast associated with signs of inflammation.

The patient, M. W—, was 60 years of age. She had noticed a swelling on the axillary side of the breast four months before I saw her. The whole of the left breast was much enlarged, and there was a dark red mottling of the skin over it, with marked œdema. This œdema was so great that it obscured the deeper parts of the breast. Extending from the outer margin of the areola

outwards to the anterior axillary line was an area of intense induration suggesting scirrhus growth, and on the inner aspect of the nipple just beyond the areola was another small hard nodule. A gland mass of considerable size could be felt in the lower part of the left axilla.

On May 14th I removed the breast with a large area of the skin over and around the growth, undermining the skin and dissecting out the subcutaneous fatty tissue as far upwards as the clavicle and as far inwards as the sternum, and removing all the sternal origin of the pectoralis major, as I thought there were a few minute nodules of growth in it. The axilla was full of enlarged glands, and these and the surrounding fatty tissue were removed. The glands did not extend up into the apex of the axilla. I was able to bring the skin together except over an area the size of the palm of the hand, which had to be left to granulate. There was some shock from the extensive operation, but it passed off in a few hours, and she has made very satisfactory progress.* The growth was typical scirrhus.

What was the cause of the wide-spread œdema and redness of the overlying skin in this case? In considering this question we must remember the position of the growth. It was situated on the axillary border of the breast, and it seems possible that in this position it may have obliterated the main venous and lymphatic vessels passing from the breast into the axilla. The large mass of axillary glands may have further pressed on other vessels which would otherwise have been able to carry on a collateral circulation. But the axillary border of the breast is not a rare position for cancer, and yet such œdema and congestion of the skin is very rare, and I do not feel quite satisfied with this explanation. If she first noticed the tumour when it started, the growth was a very rapid one, and caused early extensive infection of the glands, but often the early stage of scirrhus is so painless, that when the patient first notices the lump it may have been there some months.

In May, 1894, I had a patient sent to me with a very large cancerous growth in the breast. The skin over it was red and œdematous, and this condition of skin extended as far as the sternum on

* June 26th. She has now left the hospital, and the granulating area is slowly cicatrising: all the deep parts of the wound united by primary union.

one side and the axilla on the other. When the tumour was removed it was found that there was no infiltration of the skin by the growth, but that it was greatly stretched over it, the breast tumour being pushed forwards by a large mass of glands under the pectorals. There was no doubt as to the diagnosis before operation.

I have seen two other cases of cancer of the breast, without any suppuration, associated with what appeared to be inflammatory swelling; both were explored, and malignant disease discovered. In one the skin over the breast was of a dull red colour, and there was brawny induration in the subcutaneous tissue, with a soft fluctuating area at one spot. In the other there was marked œdema over a large cancerous growth, but no redness of the skin.

In another of the four patients who were in Guthrie Ward with cancer of the breast this month, there were two scirrhus growths in the same breast; one, on the sternal side of the nipple, had been present for twelve months, and had involved the overlying skin, the other was quite a small but typical nodule which was situated on the axillary side of the nipple, and was perfectly distinct from the larger tumour. The case was sent to me by a medical practitioner as a typical case of scirrhus, the condition of the larger growth left no doubt as to its nature before removal.

Since I gave the clinical lecture on the differential diagnosis of swellings in the breast, I have had another case which well illustrates the difficulty in distinguishing between a tense cyst and scirrhus, and in this case the difficulty was increased by the presence of two lumps in the breast. Now cysts are often multiple, but cancer very rarely. Gross has estimated that in only 3 per cent. of all cases of cancer of the breast is there more than one tumour. In this case, as in the one to which I previously referred, there were two perfectly distinct scirrhus growths. They were situated close together; one was of considerable size, the other was small.

The third case at the present time in Guthrie Ward, to which I wish to call your attention, is one of recurrent scirrhus. The primary growth was removed elsewhere more than a year ago, but it was evident from the scar that the axilla was not cleared out. Extensive recurrence had taken place in the scar, and on dissecting out the contents of

the axilla I found little masses of typically scirrhus growth scattered through the fat there. They were not enlarged glands, but quite small, intensely hard, white, radiating fragments of growth, and some were very intimately attached to the axillary vein. I tried to dissect them off, but so adherent were they that small veins passing into the main trunk through them had to be cut flush with the large trunk, and one ligature which I applied to the side of the axillary vein would not hold, but seemed to tear the opening larger. As the adherent mass passed up to the apex of the axilla and seemed equally adherent to the vein there, I divided the sternal portion of the pectoralis major, and the pectoralis minor, so as to fully expose the apex of the axilla, and then tied the axillary vein in two places and removed about two inches of the vein and adherent growth. The pectorals were then sewn together again. The œdema of the hand has not been marked; it was more apparent in the forearm, but there has been no trouble from it, and it has now nearly gone. The cephalic vein has no doubt taken on the collateral circulation satisfactorily.

I do not propose to enter into details of the operation for removal of a cancerous breast, but I wish most strongly to impress on you the fact that it must be of the most thorough kind. All incisions must be thoroughly wide of the disease, every possibly infected area (such as the pectoral fascia) must be removed, and we must not be content simply to remove enlarged glands from the axilla, but whether we feel enlarged glands there or not we must open up the axilla freely and remove all its fatty contents, in which we shall generally find infected glands, even if we could not feel them before operation. Moreover, we must remove the whole mass, consisting of the breast, surrounding fat, and the fatty tissue of the axilla, all in one piece, and be careful not to cut across the possibly infected tissue during the operation, and on no account must we use the knife, with which we have made the exploratory incision into the growth, again during the operation.

The prognosis after removal of the breast in this thorough manner, is infinitely better than it was some years ago, after the old, and what we now consider incomplete operation. It is not now simply a question of prolongation of life for some months or perhaps a year, with the certainty of recurrence. It cannot be too clearly

recognised that we have now something much better to offer to our patients. In a fair proportion of cases operated on by the thorough method the patient is cured. Some surgeons have now operated in this way for a sufficiently long period of time to show that in many cases there is no recurrence for three years, and it is generally believed that if no recurrence takes place in three years, it will not take place at all; at any rate, the number of patients who go three years without recurrence, and then get it, is very small. I have not yet written to all my patients, from whom I have removed cancerous breasts, to find out how many have had recurrence, but I know of two who have gone more than three years without any return of the growth.

The patients who have been in the hospital illustrate one fact to which it would be well to call your attention. After these extensive operations they suffer very little. Even if the skin has to be extensively removed, so that when the edges of the wound are brought together the tension is very great, as a rule their only complaint is of the constrained position of the arm. Of course in an elderly person there may be a certain amount of shock from so large an operation (and even this is not common), but if the skin can be brought together we almost invariably get primary union without suppuration, and consequently without pyrexia, and the risk of this extensive operation is very small. The well-being of the patient depends largely on our care in the details of our antiseptic method. If a large area has to be left to granulate, or to be subsequently grafted, I expect to get suppuration from its surface; but the deep parts of the wound heal without, and no rise of temperature occurs. The operation for thorough removal of the breast and axillary contents is one of the longest in surgery, and yet the distress to the patient, or I might even say the discomfort to the patient, after it, is surprisingly small. When they begin to get about again, and to use the arm, they are sure to find its movements much hampered by the axillary scar, but in time it gets free again, and they can generally use it for every purpose. I always tell patients they must expect this before the operation, but that it is a small price to pay for the chance of cure of the disease.

HYSTERICAL APHONIA.

We are indebted to the 'Medical Record' (July 17th, 1897) for the following original article on the above subject

By SANGER BROWN, M.D.

My apology for calling attention to a mere symptom is that hysteria presents such an endless variety of symptoms that one can hardly attempt to take them altogether within the compass of one short paper. I invite attention to hysterical aphonia because, while it is not one of the most frequent symptoms of hysteria, it is one of the most conspicuous when present; and though in a large majority of the cases no very great difficulty is met with in attempting to diagnose it, yet there are cases which have baffled the general practitioner successfully for a number of years. I hope I may be excused if I briefly discuss this symptom somewhat as I would do if it were regarded as a disease, because I am accustomed to discuss medical topics in somewhat of a stereotyped way.

Hysterical aphonia has been pretty clearly recognised and described for a century at least; its ætiological conditions are practically the same, of course, as are those of the disease of which it is a symptom, namely, hysteria.

In quite an extensive search of the literature of the subject, the youngest case that I have encountered was one occurring in a girl of nine, while the age of the oldest was that of a woman of seventy-four years.

In regard to the symptomatology and ætiology, at least two fairly distinct types are found; first may be considered that type in which aphonia is merely an accompaniment of many other pronounced stigmata of hysteria, such as hysterical pains, hemianæsthesia, vomiting, &c., occurring either with or without any apparent exciting cause. In such cases it frequently happens that the aphonia is not entirely pure; that is, for hours together, when the other symptoms are most complained of, the patient may be unable to raise the voice above a whisper, but in the intervals may be able faintly to phonate now and then a word or syllable. The second or pure form of aphonia, however, is that in which this symptom occurs suddenly with or without an exciting cause, continues for a longer or shorter time, and constitutes the sole evidence of hysteria.

In the impure type the aphonia may be among the first symptoms to appear, or it may show itself only after other symptoms have been present for weeks or even months. It may commence as a transient hoarseness, worse when the other symptoms are worse, or as hoarseness associated with an ordinary cold; finally, complete or almost complete aphonia supervenes, which may last from several days to several weeks or even months.

In the pure type, as already stated, the aphonia usually develops suddenly, with or without exciting cause. For instance, the patient comes down to breakfast in his usual health and spirits, and finds, much to his surprise, that he cannot raise his voice above a whisper, or very rarely he may be entirely mute; or the symptom may develop suddenly as the result of a severe emotional shock. The influence of an emotional shock will vary directly with the susceptibility of the individual's nervous system at the time of receiving the shock. This point is of the utmost importance in estimating the influence of emotion in producing disturbance of any function of the nervous system.

Many of these cases recover spontaneously and even suddenly after a few weeks or months, with or without treatment; others remain uninfluenced by treatment, the symptom persisting steadily for years. To be sure there are many mixed cases.

A great many methods have been enthusiastically put forward as successful in the treatment, more especially of the pure types above referred to; but in the last few years it has been pretty clearly demonstrated that they owed their success entirely to the influence of the suggestion with which they were accompanied; and in my opinion any method depends for its success upon the facility which it affords the patient for concentrating his efforts upon an attempt to phonate. Hypnotism has been successful in a number of instances, but not more so than the various forms of electricity, more particularly faradism, applied to the larynx, sometimes by a peculiarly shaped electrode applied internally, and at other times simply applied externally.

A method advocated by Oliver a few years ago attracted considerable attention and became known as his method, and has given excellent results. His plan was to pinch the posterior part of the arytenoid cartilages between the thumb and index finger, and thus produce an approximation of the

vocal cords, at the same time vigorously shaking the larynx and calling upon the patient to make an attempt to phonate, assuring him positively of his ability to do so. At first only vowel sounds were attempted, and gradually the pressure and shaking were diminished, until the patient was able to phonate without assistance. In case any particular sound was not satisfactorily produced, the pressure and shaking were reapplied.

A very ingenious and successful method consists in first getting the patient to cough, which in nearly every case can be accomplished; having done this, then have him cough and at the same time pronounce the different vowel sounds, and thus convince him of his ability to phonate. It is probable that in all pure cases any of these methods, if applied with suitable suggestion on the part of the operator, would be successful; but in the cases in which the aphonia is associated with other marked symptoms of hysteria, it is doubtful if complete and lasting success will be attained until the other symptoms have in a great measure subsided, and to this end it is often necessary to improve the patient's general health.

I will now describe some cases which fairly well illustrate the different types which I have alluded to above.

As representing the first type, I will quote the case of a policeman, æt. 40, of good habits, robust physique; his family and personal history are good, and he could not fairly be regarded as a man of nervous temperament. Though he received some quite severe flesh wounds in the Haymarket riot, in the main his duties have not been severe, neither have his personal or family relations been of such a nature as to cause him much anxiety. About three weeks before admission to the hospital, while travelling his beat, he felt a peculiar sensation, something like numbness, but difficult to describe to his satisfaction, commence in the radial side of the hand, extend to the thumb and index finger, and thence at times shoot up the shoulder. He continued his work until about five days prior to admission, when he suffered frequent paroxysms of severe pains in the left side and chest, accompanied by nausea and vomiting. During these attacks he could not speak above a whisper, and during the intervals he was very hoarse. Finally, when admitted he was pretty constantly and completely aphonic, though occa-

sionally a syllable would be faintly phonated. Movement of the legs was normal, knee-jerks were very lively indeed, and there was severe general jerk of the body when the patellar tendon was tapped; vision and the visual fields were normal, but there was complete absence of pain reaction to pin pricks and pinching over the entire left half of the body, including the tongue, gums, and inner surface of the cheeks, while sensation in the right half of the body was normal. Positively assured that a strong current of faradism would restore his voice and relieve his pain and vomiting, after the first application he phonated clearly, and was for the time entirely relieved of the pains in the chest and nausea. After a few daily applications he said he felt entirely well, with the exception that occasionally he had slight pain through the chest and still a little numbness in the radial side of the hand. He returned to the hospital several times for treatment after resuming his duties, but in the course of two or three weeks from his admission he had entirely recovered. This, then, was a case of an impure hysterical aphonia occurring in connection with other well-marked symptoms of hysteria, without any apparent exciting cause.

The next case is that of a woman 31 years of age, the wife of a professional man. She has had one healthy child, has correct habits, a good family history, and had always enjoyed excellent health up to two years ago, when a railway train upon which she was a passenger ran into a culvert while going at a high rate of speed, and was stopped so suddenly that all the seats were torn loose and bunched in the forward end of the car. The patient was quite severely bruised on the posterior aspect of the left hip and thigh, and received several slighter bruises on various parts of her person. No one was killed, or in fact more severely injured than herself, so the mental shock was only such as was incident to the sudden confusion and temporary anxiety for the welfare of her child, who was with her but sustained no injury. Almost immediately after getting out of the car she felt weak and dizzy, and vomited. The accident occurred at about 1 p.m., and a few hours later she again boarded a train without assistance and rode several hours till she reached her destination. She had in the meantime suffered intense and increasing pain in the legs, and had been able to walk only by putting forth a great effort. She slept several hours after a full

dose of morphine, but when she awoke the pain in her legs was as severe as ever; she felt greatly prostrated, was unable to stand both on account of pain and weakness in the legs, and was unable to speak above a whisper. She continued in this condition for two weeks, when she was seized with severe hysterical convulsions lasting several hours, with unconsciousness and opisthotonos. It was six months before she could walk without support, and about six weeks before she spoke above a whisper. Her recovery from aphonia was not then sudden and complete; at first only a word or syllable was phonated, the remainder of her speech being whispered; then she gradually improved, so that her voice only sank to a partial or complete whisper when she was tired. She had suffered many attacks of complete or partial aphonia, always associated with pain and weakness in the legs, and lasting from a few days to a few weeks, between the date of the accident and my examination several months ago. At that time she had been suffering several days from an exacerbation of symptoms like those already described, which she thought had been brought on by overwork and taking cold. For several weeks previous to this exacerbation she had been better than at any other time since the accident, was comparatively free from pain, could walk alone in the street, and her voice was comparatively clear and strong.

When examined she was in bed, complaining of pain in the legs, back, and head; of vertigo and nausea on movement, and inability to walk. She conversed entirely in whispers at first, but later, when her interest became aroused, now and then a word or two were phonated weakly and hoarsely. She said she felt no pain, and showed no signs of feeling any when pricked with a pin ever so deeply or pinched in any part of her body. The field of vision for white was reduced to the fixation point. The knee-jerks were very lively, and when the tendon was tapped the whole body responded with a violent jerk. The body was well formed and well nourished, loss of appetite and nausea notwithstanding. In bed the arms and legs could be moved voluntarily in any direction, though she declared she was entirely unable to walk, both on account of pain and weakness. I saw this patient only once and then in consultation, and cannot say anything regarding the results of treatment, but it illustrates a type in which an impure form

of aphonia is associated with very marked symptoms of hysteria developed by an exciting cause.

The next case may be regarded as illustrating the most common type of pure hysterical aphonia, not associated with any other hysterical stigmata. Miss A. A—, æt. 29, attendant in hospital for insane; nervous temperament, very competent, good general health. She had been employed several months in convalescent ward, and was not under a strain of any kind, when on rising one morning after sleeping well and feeling in her usual health, she found she could only whisper. She declined treatment and the attack lasted five weeks without mitigation, when it suddenly and permanently disappeared, the patient having attended to her work as usual in the meantime and remained in her good general health. She had previously suffered two similar attacks at intervals of several years, from which she had recovered spontaneously, and for which she could assign no cause.

The next case is that of a young man æt. 20, farmer's son, intelligent, industrious, of correct habits, fond of company, and not notably nervous. His family history is good, and he has always enjoyed excellent health, rarely having even a cold. When he was eleven years of age his father called him as usual one morning to rise, but for some reason he went to sleep again, so that his father called him a second time, speaking somewhat sharply. From that moment until he entered my office, nine years later, according to his own testimony and that of his family and numerous friends and acquaintances, he had never uttered a sound of any kind; in fact, had been absolutely mute. His playmates, when he was still a child, would throw him down and tickle him, trying to make him laugh; his face on such occasions would undergo the usual contortions, but no sound was emitted. On still more careful inquiry, it appears that occasionally a very slight sound had been emitted when he was in the act of clearing his throat, but so far as I could learn he had never been heard to cough so that he could be heard more than a few feet distant, and some members of the family in which he had lived for years were positive that they never heard him utter a sound of any kind; his communications were all made by writing. His hearing was quite acute. Movement, the reflexes, the visual fields, vision, and sensation were all entirely normal.

I had a laryngoscopic examination made by my distinguished colleague, Professor E. Fletcher Ingals, who succeeded in getting a satisfactory view of the vocal cords only after the use of cocaine; they were found to be normal in every respect, and in making the manipulations necessary to secure a satisfactory examination the patient coughed slightly. After thoroughly arousing his interest and attention by a rather minute and spirited dissertation upon the mechanism of speech (which of course he could not comprehend, but which convinced him none the less of my great skill), I assured him, with as much dramatic force as I was able to assume, that I could cure him entirely by the use of electricity, and very speedily too. I then proceeded to apply a strong faradic current to the larynx, only for a few moments, by placing a disc-shaped electrode, about one and a half inches in diameter, on each side of the organ, assuring him beforehand that after I had done this he could phonate the vowel sounds, and that as these were the basis of articulate speech, it would be necessary for him to learn to phonate them first in regaining his ability to speak. Immediately after this procedure he was able to phonate the vowel sound "e" after me; to be sure, it was very weak, nevertheless distinct; whereupon I terminated the *séance*, assuring him that the victory had been won. After this I gave him a daily treatment, and the progress was very rapid. He was soon convinced that if he said "e" he could say "eat," and if he said "o" he could say "go," and so on; in less than a week he could carry on ordinary conversation in rather a low tone of voice. He was then assured that in the course of a week more his voice would gradually strengthen, until it would finally be as strong as that of the ordinary individual, and this he found to be the case. This was six months ago, and he has continued well ever since.

This case deserves some comment on account of the youth and sex, perhaps, of the individual in whom it occurred, but more particularly on account of nine years' duration of unbroken mutism. He had seen a great many practitioners, none of whom, so far as I can learn, had made a correct diagnosis, probably because it was so difficult to get a satisfactory view of the vocal cords, and after the case had lasted two or three years without interruption a practitioner might naturally assume

that it was not one of hysterical aphonia; but really, with the history of the onset that I was able to get, the excellent state of general health ever since, and especially when the vocal cords were found to be entirely normal, there was no difficulty in making the diagnosis.

A somewhat careful examination of the literature has not enabled me to find a case that was anything nearly parallel to this in point of degree or duration. I found several cases of hysterical mutism which had lasted for several weeks, and one—that of a young woman of twenty—which had begun as simple aphonia and continued as such for several months, when it lapsed into a condition of mutism likewise lasting several months, and which finally recovered by suggestive treatment. In my opinion the efficacy of the treatment in my case was due entirely to suggestion.

The pathology of the disorder is, of course, the same as that of the other manifestations of hysteria. It is hypothetical, but most pathologists are substantially agreed upon the hypothesis, which is this: The parts of the cerebral cortex which normally preside over the various disordered functions become inactive, to the extent that they no longer respond to the behests of the will as before; accordingly, in aphonia the cortical centres from which in health the motor impulse proceeds to the muscles concerned in phonation are no longer excited to activity by the volition of the patient.

Within the last year or two Lepine and Duval—each claims priority by several months—have elaborated a hypothesis to the effect that neurons, when in a state of functional activity or potentiality, are expanded so as to be in physiological contact with such other neurons as properly participate in any particular function. During rational sleep or hysterical paralysis they are contracted, and physiological contact is broken. This theory assumes that the neurons, which are in fact protoplasmic cells, undergo amœboid movements, and experiments have been made upon frogs which appear to demonstrate the possibility of such movements on the part of neurons.

The effect of suggestion in the treatment of hysteria according to this theory might be rationally accounted for by assuming that it enabled the patient to exert an extraordinary amount of will power, resulting in the necessary expansion and contact of the neurons concerned.

NOTES.

Paralysis of the Forearm from Bicycling.

—Dr. Destot has published in the 'Gazette des Hôpitaux' an account of his own experiences. An abstract of the paper appears in a recently published number of the 'Neurologisches Centralblatt.' After a long ride he experienced paræsthesia in the fourth and fifth fingers, with impaired sensibility and paresis in the interossei lumbricales and the adductor pollicis. The paresis was followed by distinct atrophy in the affected muscles. He considers the affection to be the result of pressure upon the branches of the ulnar nerve, aggravated doubtless by the vibration occasioned by bad roads. He also considers that predisposing factors existed in the softness of the skin of the hand, and in the exhaustion of the muscles and the consequent loss of protection to the nerves lying in or under them.—*Journ. Amer. Med. Association*, July 10th, 1897.

A Röntgen Ray Examination in a Case of Asthma.

—Dr. Lewy-Dorn relates the case of a woman, 28 years old, who had dry bronchitis with consecutive pulmonary emphysema and asthma, in which, on account of the state of the lungs, a satisfactory examination as to the size of the neighbouring organs could not be made in the ordinary way. The Röntgen rays were therefore employed, and during the examination an asthmatic attack occurred, but it did not interfere with the observation. The left half of the diaphragm was observed to fall rapidly and rise slowly at each respiration, while the right half of the muscle was altogether motionless. This spectacle continued for several minutes, during which time the peculiar rough breathing could be heard distinctly. Then the patient began to cough, and the right as well as the left half of the diaphragm made deep inspiratory and expiratory movements; thick mucus was expelled, and the attack was over. The case, therefore, was one of unilateral asthma, and this the author connects with the fact that the bronchial trouble was confined to one lung.

New York Medical Journal, June 12th, 1897.

The Duration of Infection in Whooping-cough.—Weill, who, in 1894 expressed the

opinion that whooping-cough is contagious only during the premonitory catarrhal stage, has since put his opinion to the test. On various occasions he permitted nearly one hundred young children, who had not previously suffered from whooping-cough, to be associated in the same ward, for twenty days or more, with children suffering from the disease during the stage of whooping. In only one case was the disease contracted, and in this instance the patient from whom the infection was derived was in the very earliest period of the whooping stage. In three small epidemics Weill was able to satisfy himself that infection was contracted from children who had not yet begun to whoop. He concludes that infection ceases very soon after the characteristic whoop commences, and that, therefore, in a family it is not the patient who is already whooping, but his brothers and sisters who have not previously had whooping-cough, who ought to be isolated.

Lyon Méd., May 19th, 1897.

Endometritis.—Winckel ('Wiener med. Woch.') discussed this disease at the June meeting of the German Gynæcological Congress. Simple uterine catarrh usually results from distinct venous congestion. It frequently arises from this cause during infancy from improper clothing, especially tight bandaging of the body. Catarrh may also arise in childhood from want of cleanliness, irritation of the vulva and entrance of worms into the vagina. Anæmia and other diseases of the blood cause catarrh in childhood. In adult life the causes of the same disease are innumerable. After bad burns, hæmorrhagic endometritis is frequent. In acute infectious diseases this disease may arise either from entrance of the specific germ into the endometrium, or from irritation of that membrane due to chemical products evolved by the germ. Endometritis decidua polyposa results from retained relics of decidua after abortion and premature labour. In endometritis exfoliativa there is never any infiltration, as has been asserted. Between the shedding of one membrane and the development of the next there may not be the least trace of any discharge. Tuberculous endometritis is rare, the tube being more commonly the seat of tuberculous disease when it attacks the female genitals. Gonorrhœal endometritis is very frequent, and Winckel contends that the gonococcus

travels not only along the endometrium to the tubes, but also through the uterine wall to the peritoneum. Recent literature records no instance of true diphtheritic endometritis.

Indian Lancet, July 1st, 1897.

Borax in Epilepsy.—Drs. G. Angelucci and A. Pieraccini ('La Sperimentale'), after an extensive use of borax for three years in the treatment of epilepsy where careful record of the number and intensity of the attacks was kept before, during, and after treatment, have come to the conclusion that it is a very useful remedy in essential epilepsy, reducing the frequency of the seizures, sometimes suspending them entirely or substituting for the convulsions slight vertiginous attacks, or "spells" of absence of mind. It rarely is inactive. It often moderates the character of the epileptic, rendering him more tractable and calmer. Its use should not be too prolonged, in general not over three months, and the dose should not exceed six grams a day, as it may give rise to grave stupor from which one recovers with difficulty. When signs of saturation of the system, as cutaneous eruptions, acne and eczema, desquamation of the skin, and falling out of the hair, mucous vomiting, pronounced pallor of the forehead, and a tendency to corpulency appear, it should be immediately suspended. It is best given dissolved in warm water with a little syrup of orange three times a day, immediately after meals, commencing with sixty centigrams and then increasing to one gram, one gram and twenty centigrams, and later reaching to two grams a day. Thus administered it will not cause disturbances of the stomach and intestines as when given in one dose at one time in the morning on an empty stomach. It also has the advantage of being inexpensive.—*Indian Lancet*.

Koch's New Tuberculin.—In M. Nocard's report on Koch's new tuberculin to the Paris Medical Academy, the Commission of Serums makes an exception in favour of the tuberculin on account of the scientific reputation of M. Koch and the impatience of sufferers to benefit by the proposed cure, and advises the Academy to permit its temporary use. If the usual custom of the Academy were followed the new tuberculin would not be accepted until clinical proof, or at least experiments, proved the assertions made in its behalf.—*Medical News*, July 24th, 1897.

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ACUTE CROUPOUS PNEUMONIA IN CHILDREN.

BY

W. S. CHURCH, M.D.,

Physician to St. Bartholomew's Hospital.

FEW of the acute specific diseases are more easily recognised at their commencement than acute lobar or croupous pneumonia when it occurs in previously healthy adults. Its invasion is sudden, and in a very large preponderance of instances a rigor is the first intimation to the patient that he is seriously ill. With the rigor we find a high condition of pyrexia, and very frequently concomitant pain in the side and a short, hacking cough. It is not my intention to go through the other signs and symptoms of pneumonia in adults, such as the blood-streaked sputum which frequently manifests itself early in the course of the disease, the rapid pulse, the prostration, &c., or to mention the conditions which in the adult, and still more frequently in the aged, mask or modify these early symptoms. It is sufficient for my purpose to-day to remind you that the onset of pneumonia in adults is usually of such a character that we experience little difficulty in making our diagnosis.

My object in this lecture is to bring to your notice the differences which we so frequently meet with when this disease attacks young children from birth up to eight or ten years of age.

You have lately had the opportunity of seeing a good many instances of it in children in my wards, and several have very well exemplified the diversity of symptoms which they present and the difficulties of diagnosis which are met with in them.

Acute croupous pneumonia is not very common in quite young infants, but you saw an instance of it a short time ago in a baby of a few months old; from the age of two or three upwards it is

comparatively common. Occasionally in children, as in adults, an initiatory rigor occurs, and in rare instances an attack of convulsions seems to take the place of the rigor; but rigors are undoubtedly exceptional in children. In children, as in adults, the disease commences suddenly: pain in the head and belly, accompanied by vomiting, are among the earliest symptoms met with in children; vomiting, according to my experience, is one of the most constant of the early symptoms of pneumonia. Pain in the belly is much more commonly complained of by children than is pain in the chest. Diarrhœa also is not infrequent, and, on the other hand, you may have distension of the abdomen and constipation. A few years ago, a girl of about twelve years of age was admitted to my wards in a condition that closely resembled that of obstruction of the bowels; there was great distension of the abdomen, pain chiefly referred to one side, and constant vomiting. Mr. Walsham saw the case with me, and we decided to wait a little and watch her symptoms before he undertook an exploratory operation. The next day, although the abdominal symptoms still persisted, there were well-marked signs of pneumonia in the lungs, and gradually the pain in the belly improved and the vomiting ceased, and the girl made a perfect recovery.

Cough in children is frequently absent and is rarely a marked feature, when present it is unaccompanied by expectoration, such phlegm as is coughed up from the lungs being invariably swallowed. The pyrexia is high in children and adults alike, and the temperature, even on the first day of illness, runs up to a very high point, reaching 104° or upwards not infrequently. Together with the pyrexia, vomiting and headache, delirium, or more usually drowsiness, may be present, and lead to the error of thinking that you have a case of meningitis rather than pneumonia to deal with. This error is all the more likely to occur from the fact that the most careful examination of the respiratory sounds and of the chest resonance reveals no physical signs of disease. The physical signs of consolidation of the lung are often delayed

in children for several days, and occasionally are not present until the crisis has been passed. We have now in the wards an instance of the difficulty that sometimes exists in discriminating between this condition and meningitis.

Charles K—, æt. 9, was admitted to the surgery ward, April 26th, late in the evening. He was excessively drowsy, and could hardly be roused. There was no paralysis of the arms, legs, or face; his eyes were natural; he had occasional twitching of the left fingers and arm. Nothing abnormal was discovered in his chest. The respirations were 28 per minute, pulse 140, and temperature 102.8° .

The history obtained from the mother was that he was quite well until the evening of the 25th, when he was knocked down by his playfellows, and fell, striking the left side of his head against the kerb. After this he went home, and went to bed without complaining of anything. The following morning, the 26th, he complained of headache, and was extremely drowsy; this condition continued all day. During the afternoon he had frequent retching, but was not absolutely sick. Convulsive twitching was noticed in the left arm. His friends became alarmed, and brought him to the hospital late in the evening.

During the night of his admission he vomited frequently, but slept well between the attacks of vomiting. In the morning he appeared less drowsy, but still complained of headache. I saw him first at 1.30 on that day, April 27th. He was then less drowsy, but it was still difficult to get him to answer questions. He was a well-nourished, healthy-looking lad, his cheeks flushed, lips a good colour. Tongue dry, furred on the dorsum. His bowels had not acted since admission. His urine contained no albumen.

His temperature, which early in the morning had been 103.4° , had fallen at the time of my visit to 99.8° . The respirations were 36 per minute, and the pulse 140. He still complained of headache. He was ordered three grains of calomel, and an ice-bag to the head. Careful examination revealed no morbid signs within the chest, excepting that the respirations were a little hurried.

The following day, the 28th, his temperature in the morning was 100.6° , the respirations only 28, and the pulse 120. The bowels had not acted after the calomel, and he had been frequently sick

in the night. A small patch of herpes had appeared at the left angle of the mouth, percussion and auscultation of the chest gave no evidence of anything amiss with the lungs, but he complained of pain in the left side. I need hardly trouble you with the daily notes of the case, suffice it to say that the next day, the 29th, the fourth day of the pneumonia, the physical signs of impaired percussion below the level of second rib in front and all over the left side behind appeared, together with some fine crepitation and harsh breathing. His fever had been high during the night, accompanied by some delirium, and the temperature during the day did not fall below 103° , and in the afternoon was as high as 104.4° . It was not until May 3rd, the eighth day of his pneumonia, that the physical signs attained their maximum, when there was marked impairment of percussion over the whole of the left lung posteriorly, less marked anteriorly, together with well-marked bronchial breathing, bronchophony and crepitation. On this day the temperature fell, although the boy had not a decided crisis; for in addition to the pneumonic condition of the left lung, a small portion at the base of the right lung became affected, and kept the temperature above the normal until the tenth day of the illness.

Notwithstanding the difficulties that I have already mentioned, a careful consideration of all the features presented by a case generally enables one to feel pretty sure that you have pneumonia to deal with rather than the invasion stage of one of the other acute specific disorders. Among your safest guides are a careful study of the respiration and pulse. The rapidity of the respiratory act and the dilating nostrils of the child may raise our suspicions, even when the leading symptoms point rather to gastric or cerebral disease. In pneumonia in children the breathing is not only accelerated, but it is often characteristic of the disease, each expiration being accompanied by a grunting noise; attention has been called to this of late by Professor Hensch in his work on the 'Diseases of Children,' but it was observed, and the value of this symptom recognised in this country years ago. You know that in health the frequency of the pulse to that of the respiratory act is as 4 or $4\frac{1}{2}$ to 1, and that this proportion is fairly maintained in most pyrexial conditions, but that is not the case in pneumonia, the rapidity of the breathing is often out of all

proportion to the pulse, the respirations running up to 60, 70, and more a minute, whilst the pulse may not exceed 120 or so. Scarlatina, like pneumonia, is an exception to the ordinary rule, as at the outset of that disease we often meet with a remarkable increase in the rapidity of the pulse without a corresponding rise in the number of respirations per minute, scarlet fever being thus the converse of pneumonia so far as these symptoms are concerned.

When dealing with pneumonia in children it is not so much the absence of symptoms commonly met with in adults, such as pain in the side, blood-stained expectoration, evidence of consolidation in the lung, &c., which is likely to mislead one, as the presence of others which we do not usually associate with pulmonary disease. Thus, as I have already mentioned, you meet with instances in which the severe and the constant pain in the head, together with the vomiting, closely resembles that present in meningitis. So close, indeed, is this resemblance, that Professor Henoch says, "it always appears to me that many of the cases of recovery from meningitis, especially from tubercular meningitis, have been nothing but cases of pneumonia with cerebral symptoms which were wrongly diagnosed."

You may fairly ask, what is the cause of this difference in the symptoms of the disease in young children? I think that the explanation lies in the greater impressibility of the nervous system in young subjects. The drowsiness, stupor, and delirium which occur so early and are such marked features in children all point to the central nervous system, and we know how easily this is acted on by various poisons, such as opium, alcohol, &c., in the child. What it is which thus acts on the nervous system is not, I think, yet fully shown. Is it merely the rapid increase in the temperature of the body which takes place? or is it due to the action of some toxin produced by the pneumococci which we know are always present in large numbers in this disease? We meet with as high a range of temperature in other diseases without these marked cerebral symptoms, so that I incline to the opinion that it is dependent on something other than mere temperature. We must at present admit that we do not know, although we may hope soon to have fuller information on this most important point, for already those who are working in the interest-

ing field of bacteriological pathology are ready to furnish us with an antitoxin for pneumonia, and this leads us to say in conclusion a few words on the treatment of this condition.

Fortunately in childhood, as in adult life, the tendency of acute croupous pneumonia is to recovery; and in children, provided that they are sound in other respects, death from uncomplicated croupous pneumonia is a very rare event. When death does occur it is nearly always due either to some unsoundness which existed before the pneumonia, or to the general infection of the system by the pneumococci secondary to the affection of the lungs. The commonest and most fatal of these is an acute cerebro-spinal meningitis, which is associated with and, I think, undoubtedly due to the presence of the pneumococci on the meninges; this condition you may remember to have seen a few days ago in an infant who died in my wards; occasionally the connection is not so direct, and purulent otitis apparently dependent on the pneumococci is followed by thrombosis of the lateral sinus and either meningitis or cerebral abscess. At other times the inflammatory condition spreads to the pleura or pericardium, and purulent pericarditis and pleurisy occur.

The tendency of pneumonia towards recovery is so decided that our treatment should not be too active. Repeated bleedings, blisterings, and semi-poisoning with tartar emetic have fortunately long ceased to be the routine treatment; nevertheless, I still think we must be careful in dealing with children that our treatment is free from any suspicion of harm. The treatment of pneumonia has for years been one of the battlefields of therapeutists. Heroic bleedings, nauseating doses of tartar emetic, and more recently digitalis, cold bathing, quinine, aconite, and a number of other antipyretic drugs have been extolled by their respective advocates, and most contradictory statistics published of their value. I believe the only reasonable conclusion to come to is that none of them really exercise any influence on the disease itself, and that our business is to most carefully watch the symptoms, especially the condition of the pulse, respiration, and skin, and to make use of such means as we deem advisable for alleviating the most prominent symptoms; whilst at the same time we are careful to avoid using drugs in such quantities or of such powerful characters that we

may, in our attempts to do good, on the contrary harm our patients; and this is especially to be remembered in the case of children.

When the vomiting is excessive and the bowels constipated, I think that relief is obtained by frequently repeated small doses of calomel until the bowels have been freely moved; and I am quite sure that the restlessness and discomfort is greatly mitigated when the skin acts well, and with this purpose I am in the habit of ordering full doses of diaphoretics, and believe that none act better than the old-fashioned acetate of ammonia, ʒj of the Liq. Am. Acetatis diluted with an equal portion of water, and given every three or four hours to a child of two or three years of age, the dose being increased in proportion to the age of the patient. I avoid as much as possible giving antipyretics such as antipyrine and phenacetine. If given in sufficient quantities to have a sensible effect on the temperature they are apt to be followed by depression; but when there is much restlessness, with high temperature, a few grains of one or the other of these drugs sometimes act very well.

It is not often that children complain of pain in the chest in pneumonia; when they do I usually, unless the weather is very hot, apply a poultice or warm compress; the use of ice compresses has been much extolled in pneumonia, and I have often tried it both in adults and children; some adults, especially in hot summer weather, like it and find relief from its application, although I have not found that it has influenced the course of the disease in any way. Children invariably in my experience resent the application of ice compresses.

The thirst which is usually present causes children in most instances to take willingly sufficient nourishment in the shape of milk and water or milk and lime water; whey is tolerated by the stomach better than milk in some cases, and trial can be made of it for four-and-twenty hours or so. In some cases of delirium and stupor it is impossible to get children to take food voluntarily, and nasal feeding has to be adopted, but this is very seldom required for more than twenty-four or forty-eight hours.

It is only in rare instances that stimulants are required by young children before the crisis has taken place; after its occurrence an ounce or so of wine in the course of the day appears in severe

cases to favour convalescence. Occasionally, even in quite young children, alcoholic stimulants are required from an early period, especially in those cases in which vomiting, or more rarely diarrhoea, has reduced the strength of the little patients at the very commencement of the disease.

In conclusion, let me impress on you that although I believe not much can be effected in the way of controlling the disease by direct treatment, there are few if any conditions which call for closer watching or greater care, and that a timely assistance given to nature at the right moment often makes the difference between life and death.

CLINICAL LECTURES ON URINE.

Delivered at University College Hospital by

J. ROSE BRADFORD, M.D., F.R.S., F.R.C.P.,

Physician to University College Hospital.

No. I.

THERE are but few diseases which do not alter in some way the composition of the urine, and there are several diseases which can only be diagnosed by an examination of the urine,—thus diabetes mellitus may be overlooked unless the urine is examined as a routine measure, and in the same way a contracted or granular kidney may be overlooked. As regards the examination, though much that is recommended is more applicable to hospital than private practice, still as near an approach to hospital practice as possible should at any rate be attempted. The first rule, especially in insurance cases for instance, is that you ought to see the patient pass the urine yourself. In making your patient pass the urine for examination you should follow the rule of the surgeons and cause it to be passed in two portions, one small quantity first to wash out the urethra, and then a larger quantity; this is more especially necessary when the urine contains pus or blood. In women it is frequently important to pass a catheter in order to make certain whether pus cells, blood, &c., come from the urinary tract or not. The use of the catheter may be necessary in the male in cases of supposed malingering. In many diseases it is absolutely necessary to take the

whole twenty-four hours' urine and to mix it up. I am perfectly well aware that it is very difficult to do this in private practice, but it is well to try and obtain this. It is important in several renal diseases, for example diabetes; in granular kidneys it is of consequence because of the determination of the specific gravity. Plenty of people may pass urine of 1005 specific gravity temporarily, but a specific gravity of even 1010 for the whole twenty-four hours' urine would be very abnormal. Hence you should be very careful to examine a sample of the whole twenty-four hours, and not a particular sample that the patient may bring you at the time you see him. There is another important point to attend to in this matter, and that is to obtain if possible a specimen of the starving urine—the urine passed before breakfast,—and also another specimen of the urine passed two or three hours after the heaviest meal of the day, lunch or dinner, as the case may be. This is important, for instance, in some cases of gravel, because in such patients the gravel tends to deposit in the morning urine before breakfast and in some cases of albuminuria and glycosuria, the abnormal substances are found only after meals. For an ideal examination of the urine you should have, then, the whole amount passed during the twenty-four hours mixed up, and take a sample of that; you should take a sample of the starving urine and one also of the urine passed after the heaviest meal of the day. If you wish to make perfectly certain that albumen and sugar are not in the urine, this plan must be adopted.

In disease the urine may undergo qualitative or quantitative changes, or both. It is commonly stated that the average quantity of urine passed in the twenty-four hours is some fifty ounces, and that it varies from a minimum of twenty ounces to a maximum of eighty ounces. A more important clinical point is that the amount of water lost is about 50 per cent. of the fluid taken in; that is to say, four to five pints is the usual amount for an adult to take in the day. I am not going into the question of the physiology of the urinary secretion, but I would say in reference to the water of the urine that it depends practically upon the rate of the blood-flow through the kidneys, and upon the activity of the glomerular epithelium, and these are the proximate causes of all variations in the amount of the urine. But more important from

a clinical point of view are the other ultimate factors on which the amount depends. Firstly, the quantity of water passed in the urine depends on the amount of fluid taken in; secondly, it depends on the amount of fluid that is lost by different channels, and more particularly by the skin, the lungs, and the bowels. In health, the amount of fluid in the tissues remains pretty constant, so that variations in the amount of fluid in the tissues do not appreciably affect the amount of urine in health. In disease, however, great variations occur in the amount of fluid in the tissues, and therefore you have in addition to the loss by the lungs, the skin, and the bowels, to take into account the varying amount of fluid in the tissues. If a patient becomes dropsical the amount of urine necessarily diminishes, and the variations in the amount in dropsy (which is a condition in which there is excess of fluid in the tissues) must necessarily affect the urine, even if the dropsy is not dependent upon renal disease. In the case of a patient with heart disease a sudden diminution in the amount of urine by no means indicates necessarily the onset of grave renal disease, it may point to the onset of dropsy. The quantity of urine depends, then, on the rate of the flow of the blood through the kidneys and the activity of the glomerular epithelium, and beyond this there are the questions of the amount of fluid ingested, and the amount lost by other channels, and the presence or absence of dropsy.

In disease the quantity of urine may be increased or diminished. Most diseases tend to diminish the quantity of urine, but there are some diseases in which the quantity of urine is increased. The increase in the quantity of urine may be temporary or more or less permanent. There are various conditions which will cause a temporary increase in the flow of urine quite apart of course from copious drinking, which is of course the most obvious cause; it is asserted that in excessive drinking you may have a specific gravity of the urine lowered to one comparable to that of water. A milk diet, containing as it does a diuretic like lactose, will lead to the excretion of a copious dilute urine. A temporary increase in the amount of the urinary water occurs on the subsidence of nervous paroxysms, *e.g.* after epileptic and hysterical attacks, and after asthma. Other instances are those in which there is a subsidence of dropsy, and the

subsidence will often show itself at first rather in an increased quantity of the urine than by any notable diminution in the amount of the dropsy. Patients with chronic Bright's disease passing an increased amount of urine are sometimes thought to be going on to the stage of cirrhotic kidney, when the increased flow is often due to a diminution in the amount or disappearance of the dropsy. A temporary increase in the flow of urine is seen as a result of the re-establishment of the urinary secretion after the flow has been obstructed. Thus in renal colic, after the calculus has passed, the patient passes much urine of a very dilute character. A temporary increase in the flow of urine is also seen during the subsidence of acute and subacute inflammation of the kidney. In many diseases like diabetes mellitus and diabetes insipidus, renal cirrhosis, and other diseases of the kidney, the quantity of urine is permanently increased. In all these conditions the quantity of urine is permanently increased as long as the disease lasts. In diabetes mellitus and in diabetes insipidus the quantity of urine is greatly increased, but the increase is usually greater in diabetes insipidus than in diabetes mellitus. In diabetes insipidus the patient may pass any quantity from ten to twenty pints, and one case is recorded in which thirty pints were passed in the day; whereas in diabetes mellitus the quantity of 150 to 200 ounces is rarely exceeded. These diseases are classified as causing a permanent increase in the urine, but in both the increased flow may stop suddenly, and more especially in diabetes insipidus. In diabetes insipidus the urine is like water, quite clear, with a low specific gravity, whereas in the other form the specific gravity of the urine is as a rule high.

In certain forms of Bright's disease the quantity of urine is greatly increased, and in all forms of acute Bright's disease a temporary increase in the amount is seen during the subsidence of the dropsy. In chronic Bright's disease the flow is often increased, provided the patient is not dropsical. In renal cirrhosis the quantity of urine passed is considerable, 80 to 100 ounces. In the above-mentioned cases of Bright's disease about 60 ounces would be an average, that is to say, a little above the normal; but in the granular kidneys the quantity is considerably above the normal, but rarely above 100 ounces. Certain forms of chronic

Bright's disease may produce a greater increase in the urinary flow, readily diagnosed, because the quantity of albumen present is considerable; in the granular kidney the urine does not always contain albumen, and a granular kidney may occasionally cause death without producing albumen in the urine.

The quantity of urine passed may be diminished, and may even be suppressed. It is customary to describe two forms of suppression, one obstructive suppression, and the other non-obstructive suppression. In obstructive suppression the suppression of the urine is caused by an obstruction to the flow of urine along the ureters. The urine may be pent up behind the obstruction, or there may be actual suppression, with no urine in the renal pelvis. These cases of obstructive suppression may, therefore, be divided into two groups; in the one the ureters and renal pelvis are dilated and distended with urine; in the other, notwithstanding the obstruction, there is little or no distension of the urinary channels. Cancer of the uterus frequently involves the ureters, and under these circumstances the ureters may be distended to the size of the index finger. One of the most frequent causes of obstructive suppression is the impaction of a stone in a ureter, when the other kidney is either absent or destroyed by previous disease. Obstructive suppression is a condition that can go on for a considerable time, and it is usually stated that it may go on for from eight to ten days. The most remarkable thing about it is that the patients are not very ill, they do not feel very ill, nor do they look very ill on casual examination. Such patients are not very ill till very shortly before their death, and they often die suddenly, and the mind may remain clear until the last. The only signs that point towards the dangerous condition of these patients are, that the pupils become contracted, the patient's temperature steadily falls, so that they have a temperature of 96° or 95°, and then just before the end they may have muscular twitches. But the main point is that in these cases of so-called obstructive suppression, where there is a mechanical obstruction, they do not suffer from what is called uræmia; they die quite suddenly, usually about the middle of the second week, and the whole illness they may be completely in possession of their faculties, without any serious symptoms of any kind.

Non-obstructive suppression is a very serious malady indeed, and is met with in a variety of conditions; it is a condition in which there is complete and generally fatal suppression, without there being any mechanical impediment to the exit of the urine. It may occur in people with perfectly healthy kidneys as a result of various operative procedures. When I say a perfectly healthy kidney I mean they do not show any signs of gross disease. Non-obstructive suppression of urine may occur from passing a catheter; it is not very uncommon for this to occur when there is serious disease of the kidneys. Complete suppression may also occur as a result of various abdominal injuries and diseases, and also sometimes as a sequel to operations. Exploratory operations on the kidneys are very liable to lead to suppression and uræmia if these organs are diseased. Fatal suppression has occurred more than once in cases where the kidney has been cut down upon for stone, and where there was in addition chronic Bright's disease or waxy disease; and I have heard of a case where fatal suppression followed nephrectomy, and where the kidneys showed no gross lesion post mortem.

Non-obstructive suppression with apparently healthy kidneys is seen sometimes in various serious abdominal diseases. In one case a patient with perforation of the duodenum died not from peritonitis,—he was walking about the wards,—but from suppression of urine. Recently there was a case of a woman who after an abortion died from suppression of urine. There was no peritonitis, and the uterus showed no signs of disease, and the kidneys showed no gross lesions although they may have been slightly fatty. As a result of severe abdominal injuries suppression is not uncommon. The next variety of non-obstructive suppression is that occurring in diphtheria. Here the mechanism is not certainly known, but it is probable that it occurs as the result of the action of the toxins produced in diphtheria on the renal epithelium.

A familiar form of complete suppression is that seen as a result of very severe congestion, active or passive, of the kidneys. Very acute nephritis produced by scarlet fever may cause suppression of urine, and this is different to the suppression occurring in diphtheria, in that the latter disease does not cause the gross lesions in the kidneys

seen as a result of scarlet fever. In scarlet fever there are obvious changes, and in some cases the congestion has been so extreme that the capsule of the kidney has been ruptured. In the passive congestion of the kidneys due to thrombosis of the renal veins, complete suppression may occur, but the thrombosis is often unilateral, and then suppression does not occur. Suppression sometimes occurs as a result of acute congestion produced by poisons, and there was a case recorded a few months ago in which a woman got severe suppression from the application of a very large blister.

Non-obstructive suppression is very rapidly fatal; the patients usually die in two to three days, with very severe vomiting, and headache, convulsions, and coma—that is to say, the clinical picture of certain forms of uræmia.

Diminution in the flow of urine is seen under a variety of conditions; first of all, it will occur if large quantities of water are lost from other channels. Thus in febrile diseases the hurried respirations, the sweating will lead to an increased loss of water from the lungs and skin, and thus the urinary flow is diminished, and a typical instance of this is seen in pneumonia. In any disease in which diarrhœa is a marked symptom there may be a considerable diminution in the quantity of urine, and if the diarrhœa is very severe, complete suppression may occur. Then, as mentioned above, the presence of dropsy will diminish the flow of urine, and this whether the diminution in the flow of the urine is the cause of the dropsy, or the onset of the dropsy is the cause of the diminution of the flow; but the fact remains that they vary inversely, there is a relation between the two of such a character that the fluctuations in the quantity of urine will very often give more reliable information of the subsidence of the dropsy than any physical signs. Anything which diminishes the rate of the blood flow through the kidney, and more particularly venous congestion, will produce diminution in the flow of the urine; thus a scanty secretion of urine occurs as the result of abdominal tumours, collections of fluid in the abdomen, and in heart and lung disease, and so forth. In all these the quantity of urine is greatly decreased, owing to the passive congestion of the kidney. In these conditions the quantity is usually not diminished to much under twenty ounces. Diminution in the flow of urine

may also occur in diseases involving and destroying the secreting structures of the kidney, the typical instance is chronic Bright's disease with dropsy; but in many diseases destroying the renal tissues a more or less abundant dilute urine is passed.

The *specific gravity* of the normal urine may vary from 1015 to 1025, and in disease the specific gravity may approximate to that of water, or rise as high as 1060. An abnormally low specific gravity is usually a sign of serious disease if it is a permanent condition, and if it is found in the whole of the twenty-four hours' urine. Urine of low specific gravity is secreted after copious drinking, and in the conditions described above, where there is a temporary increase in the urinary flow; but a permanent lowering means usually some serious kidney lesion, such as granular kidneys, waxy disease, &c., unless it is due to diabetes insipidus. As regards the increase in specific gravity it is of little importance, except in the cases of diabetes mellitus. All concentrated urines have a high specific gravity. One of the most important points about diabetes is that occasionally in this disease there is a low specific gravity, and yet there may be a considerable amount of sugar present, even with the specific gravity as low as 1010. In diabetes the specific gravity and the percentage amount of sugar do not necessarily vary together. Supposing two samples of diabetic urine, one of 1040 specific gravity and one of 1045, it does not follow that the higher specific gravity indicates the greater amount of sugar. In diabetes mellitus there is a great increase in the amount of urea in the urine; but if the patient is passing large quantities of urine, then the specific gravity does vary with the amount of sugar present, because the other urinary constituents are present in comparatively such small amounts that the sugar is the most abundant urinary constituent. If a patient with diabetes only passes very small quantities of urine the specific gravity is very much affected by the other constituents present; if the patient passes large quantities, then the other urinary constituents are scanty, and the specific gravity does vary with the sugar.

DEMONSTRATION OF CASES

AT

**The West London Hospital, Hammeramith.
March 10th, 1897,**

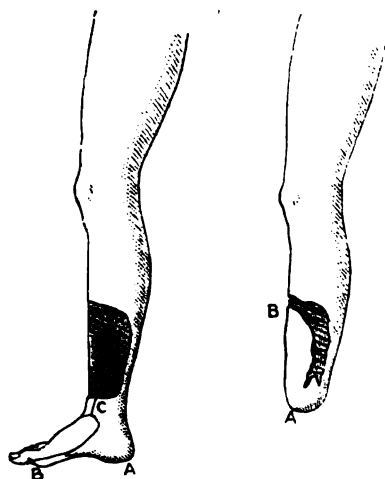
By C. R. B. KEETLEY, F.R.C.S.,

Surgeon to the Hospital.

Plastic Amputation of the Foot for Ulcer of the Leg. Cases of Inguinal Hernia—Undescended Testis—Femoral Hernia—Anchylosis of Hip—Double Anchylosis of Hip—Relapsing Appendicitis.

GENTLEMEN,—The patient who is now taking off his artificial foot had a large epitheliomatous ulcer of the leg. The ulceration went right down to the bone, and the question arose what was the best thing to do with it. First of all, the whole disease was removed very freely, and then an incision was brought down the middle of the dorsum of the foot and round the foot just above the toes. The skin and soft tissues were then turned back as a flap as far as the ankle, and all the bones of the foot were removed except most of the os calcis. All the nerves and vessels of the sole of the foot were left, and the long tendons cut away. The resulting flap was turned up to cover the ulcer, and the os calcis was wired to the tibia. That is called an osteoplastic amputation of the foot. The first operation of that kind was performed here ten or twelve years ago, but it was a very much worse ulcer, and extended almost to the knee. The man came to have an amputation done. I thought I could save his leg and leave him a stump. In this case I cut away a little more of the tibia and removed all the bones of the foot. Professor Rydiger, of Cracow, considers the preservation of a part of the os calcis a gigantic improvement; I do not think so. The kind of case for which plastic amputation of the foot is justifiable, would be a large ulcer covering the greater part of the leg, and then there is no length of limb gained by preserving the bone in the heel, because you have to cut away just as much more of the tibia in order to get enough skin, &c., to come high enough to cover the site of a large ulcer. There is possibly a little to be said in favour of preserving the os calcis, because it gives a trifling help in the nutrition of the tissues over it. The question is

exactly that of Syme's v. Pirogoff's amputation. The skin of the heel, however, does not get nutrition from the os calcis, but from the arteries and vessels which go from the tibial and plantar vessels. The great advantage of the operation I have done upon this man is that he can walk upon



Diagrams of original case before and after operation
(see 'Lancet,' Nov. 28th, 1885).

the stump, which can stand any amount of pressure. If he had had any other kind of operation he would simply have had to walk upon his knee all his life. In fact, it has all the advantage of a Syme over an amputation through the leg. You will see there are still some of the remains of the eczematous condition which led to the mischief. If he would come into the hospital for two or three weeks we could get that eczema well, but we shall take care not to let it get any worse. I have done several forms of this operation, but do not propose to take up your time by talking about them now. This is the first case in which the nature, depth, and position of the disease justified me in keeping a part of the os calcis, and in which I have utilised the skin of the dorsum as well as of the sole of the foot. Had the disease been ordinary and not malignant ulceration, amputation would not have been required at all, as I should not have had to cut away the muscles and periosteum of the front of the ankle.

I wish now to show you three typical kinds of hernia which have all been operated upon for radical cure. The first is a *strangulated inguinal hernia, congenital*; the second is a *congenital*

inguinal hernia, not strangulated, but with a retained testis; and the third is a *strangulated femoral hernia*. Always when operating upon hernia, whether for radical cure or for the relief of strangulation, I go straight down to the aponeurosis of the external oblique. That is a whitish or bluish shining membrane, and when we come to that we know where we are. After tearing aside all the fat, &c., the hernia comes out like a flesh-coloured ball, covered with the proper coverings of the hernia, which come from the external oblique,—what used to be called the external spermatic fascia. The next step is to go through the coverings at one particular point, making an incision about one inch in length; you should not go through with a dash, but should partly cut and partly tear through them, until at last at that particular spot you have got down on to the peritoneum or sac of the hernia. Now I always put two Wells forceps on the sac, and then proceed to separate the sac from its coverings which adhere to it. It does not matter very seriously if you accidentally tear up these coverings in separating them from the sac, because you do not want them for any particular purpose afterwards. In a case of oblique congenital hernia you have to separate the sac from the cord. This is sometimes a difficult matter, but there is one point at which it is most easily separated, namely, at the internal ring. Moreover, when it is strangulated the constriction is usually at the internal ring; it is best, without any delay, to slit up the anterior wall of the hernial canal and expose the internal ring. Having done that and got the sac and the cord separated, the rest of the operation is simple. You tie the sac high up, or else tighten it up with a purse-string suture. Some people put a suture through it and fix it to the abdominal wall in that way, but it is not necessary. Then with sutures you narrow the muscular and aponeurotic boundaries of the internal ring till there is only just room for the cord, and, finally, you close the cut originally made in the anterior wall of the canal. The credit for the most important steps in this mode of operating should be shared between Macewen, Lucas-Championnière, and Bassini.

The next patient had *undescended testis*. He had a similar operation to that I have described, and in addition to that his testis was brought down. In order to get the testis down it is always

necessary to cut in two the fibrous structures, which really hold the testis up. A testis which is not descended can seldom be got down to the bottom of the scrotum, but becomes indirectly attached somewhere near the crest of the pubes. I have seen a testis attached very nearly to the anterior superior iliac spine. In the present case what I suppose was the gubernaculum testis was attached close to the external inguinal ring, instead of going into the scrotum. It had to be cut freely, and then the testis came down. It is most important that the testis should come easily down, for if you drag the cord over the sharp inner edge of the internal ring the patient cannot be expected to stand it, and it might be injurious to him. Having got the testis down, the next step was to keep it there. It is customary to keep it down by something like the basket hilt of a single-stick. No person can be expected to wear a thing like that for more than a fortnight, therefore it occurred to me to fasten the testis to the fascia of the thigh, making one incision in the scrotum and another in the thigh. This patient has one suture through the tunica albuginea, and another through the testis, connecting it as indicated. The longest I have left such sutures in is five months, but there is no reason why it should not be five years, as no discomfort is felt even with the trousers on. This testis is now in position, and it is of a fair size for one so long undescended.

The next patient is thirty years of age, and was operated upon five months ago. The point about *femoral hernia* is that, just as in inguinal hernia, you cannot close up the opening, for you must leave room for the vein and artery to come through. Supposing the hernia has been put back completely, I generally bring one end of the suture which ties the neck of the sac up through the external oblique aponeurosis so as to get it out of the way. To close the whole a suture is passed right down to the ilio-pectineal line and close to the vein, making sure the vein is on the outer side, close down upon the bone, taking hold of fascia, muscle, and periosteum. Then it is passed to the front through Poupart's ligament from before backwards and out again about half an inch up. Then it is brought back and passed again through the pectineal tissues. It is then tightened up. This sutures the *front* of Poupart's ligament to the upper surface of the ilio-pectineal line. The same thing is repeated a

little further in, and that closes up the ring, and makes also a kind of valvular opening.

I would now like to show you two cases of *anchylosed hip*. The first patient has had disease of both hips. I have seen a good many of these cases, especially when I was surgeon to the Surgical Aid Society. As the patient can walk so well, you may be surprised to find both hips anchylosed. Many things are described in text-books very freely by people who have never seen the conditions. Persons with anchylosis of the hips can get along very well even if they do not move the knees on one another. The books used to say that persons with anchylosis of both hips can only walk like a pair of compasses. I have seen many such cases walk by using only the knee and the ankle; and there is no doubt that the sacro-iliac articulation moves a little, and I think the pubic may also. For anchylosis of both hips the proper thing to do is osteotomy of one hip, leaving it in a good position to anchylose again firmly, and then an excision of the other hip so as to make it moveable. It is not wise to do an excision of both hips, because a freely excised and moveable hip is always a weak hip. I propose to osteotomise both of these patients some day. I have done many operations of this kind, and I have tried all kinds of ways. Certainly one of the best, as well as the most workmanlike way is to remove a wedge of bone. In almost all these cases, if they stood with their back perfectly straight and the sound leg perfectly straight, the anchylosis is rectangular. If the pelvis is looked at from the front, it is generally slewed to one side—in other words, there is anchylosis with adduction; when the knee is pressed back the abdomen rises up. Generally speaking, the head of the bone is destroyed. In the first six cases I operated on, I simply made an incision and put in a chisel and cut the femur in two. It is not always an easy thing to do, and requires a lot of calculation, and all the soft parts are very much shortened. The condition is usually many years old, and in putting the hip straight you sometimes may feel that you are running the risk of tearing the femoral vessels. Operation by dividing the femur below the trochanters is only partial compensation for one deformity by causing another. The best operation is to take a wedge from the bone, and then cut or snap the remainder in two. It is an easy operation. There was

reason for not being in a hurry to meddle with this particular patient, for he came in with a residual abscess, which was treated by erosion and iodoform. A sinus has been left, and it is not easy to make sure that a sinus is aseptic. According to ordinary rules, it would be a shocking thing to osteotomise a boy who had so recently had an abscess; but that shows one of the advantages of wedge osteotomy. If in cutting out the wedge any tuberculous focus was discovered, no harm would be done, and the osteotomy need not necessarily be completed. If the wedge were healthy, the osteotomy would be justified and successful. This boy's deformity is exceptional, because he has got abduction instead of adduction, which does not occur once in seven times. If I had not had the patient under an anæsthetic I should not have known whether he had osseous or fibrous ankylosis. Even if it had been fibrous ankylosis I should have decided to do an osteotomy, because I have tried breaking down the adhesions, and have found that the limb tends to relapse into the old state of contraction.

I wish next to call your attention to a case of *relapsing appendicitis*. The patient is a young woman æt. 24. She has six times had symptoms of perityphlitis, and it was decided by Dr. C. Andrews and myself that something ought to be done to prevent her having these visitations. I took her into the hospital and opened her abdomen. I did not feel or see the appendix, but I found the cæcum and the ileum joining it. Whilst I was turning over the cæcum I felt that there was a very great thickening, just like a solid tumour, near its junction with the ileum. Upon reflecting the peritoneum off the thickening, I found the appendix beneath. There was a mass of cicatricial tissue, and embedded in it was the appendix. The peritoneum, quite smooth and shining, spread over everything just as though nothing was wrong. I cut a considerable opening into the cæcum in removing the appendix and margins of its opening, and the operation became practically one of partial enterectomy. To close the aperture, I first sutured the mucous membrane together, and let the knot of the suture pass into the cæcum. Then I put in a row of Lembert's sutures. Fine silk was used. The patient has done perfectly well, and is convalescent. She is now (April 6th, 1897) getting better, and getting about as usual.

CLINICAL CASES

BY

G. G. MORRICE, M.A., M.D.Cantab., M.R.C.P.,
Physician to the Salisbury Infirmary.

CASE I. *Scleroderma*.—We have had recently in the Salisbury Infirmary an example of this rather rare disease. It seems proper to point out to you in what ways he conforms to the usual type, and in what ways he illustrates special points which other observers have noticed; but I shall not attempt a general account of the malady, merely reminding you in preface that there is a group of diseases in which keloid of Addison, false keloid of scars, scleriosis, sclerema neonatorum, morphœa, hemiatrophy of the face, morphœa herpetiformis, Raynaud's disease, and other conditions are more or less intimately linked together. It was Hilton Fagge, who in his monograph in vol. xiii of the 'Guy's Hospital Reports,' first drew attention to the fact that many cases of scleroderma which had resisted all treatment in hospital ultimately recovered spontaneously.

Mr. Hutchinson has largely increased our knowledge of the subject, and in particular we owe to him the theory that hemiatrophy of the face is due to morphœa of the fifth nerve. The connection between and the peripheral distribution of nerves is worked out in two articles in the 'British Medical Journal' for 1895, on "Morphœa Herpetiformis."

Samuel B—, innkeeper, was admitted to Queensberry Ward on February 29th, 1896, suffering from dermatitis affecting both feet, legs, and lower part of thighs. It is difficult to define the condition, whether to call it superficial ulceration or eczema; certainly there was a distinct loss of substance in the true skin, for at one stage granulation tissue was exuberant; however, this rapidly healed with rest and application of "Lassar's paste." On examination the skin was found to be hard and discoloured, very closely resembling the condition so familiar to us in the dissecting room. The only part retaining its softness was a small area round the umbilicus. This condition is practically the same at the present time. He is unable to close his hands owing to the stiffness; the palms are soft, but the backs of the hands are very stiff. The arms present a fine chequered appearance, brown and white areas adjoining one another.

The forearms are more uniformly brown, but on looking closely you will see a few of the characteristic white ivory patches of morphea, some as large as a threepenny piece. There are non-pigmented areas surrounding each nipple and running up into the axillæ. I wish especially to draw attention to the white areas over the scapulæ. They are very interesting, as apparently representing the patches in the same positions figured by Mr. Hutchinson in the article on "Morphea Herpetiformis." Yet on consulting a very exhaustive German monograph, 'Die Sclerodermia,' by Drs. Lewin and Heller, I do not find this mentioned.

Now let us consider the leading features illustrated by our patient.

1. Capillaries of the cheeks. Hutchinson says of one patient, Mr. H. E—, "The cheeks, &c., showed tufts of ciliated vessels (stigmata);" and of another patient, Mrs. B—, "The cheeks showed little tufts of ciliated vessels." This description would tally with the condition present here.

2. Superficial ulceration or dermatitis of the legs. This appears to have been a recognised complication in Addison's cases of keloid, for Fagge says, "The cuticle exfoliated. The cutis became liable to undergo a superficial ulceration, with consequent scabbing." During the past winter the right leg again broke down, and the patient was admitted to the infirmary with an attack of acute eczema, which was treated with equal parts of black wash and lime water, and an ointment of carbonate of zinc.

3. Albuminuria. The urine generally contains about three quarters of albumen on boiling. This is certainly not a frequent complication in sclerodermia. Fagge mentions two cases which came under Dr. Gull's care. One had temporary albuminuria; in the other, "both hands were useless from induration of the skin and subcutaneous tissue. This state extended more or less generally over the body. The urine was at first quite normal; but as the change in the integument became more general, the water became albuminous, and continued so till death."

I think we may assume that the nephritis was primary in my case. During his attack of eczema there was slight dyspnoea, otherwise one might have supposed that we had to do with functional albuminuria.

At first sight the skin appears void of hair, but on careful inspection a very fine down can be seen. Nor are the sweat-glands atrophied, for the patient says that in the summer, when he perspires more, the skin feels softer.

As regards prognosis in the general case, 16 per cent. recover entirely, 30 per cent. improve, 29 per cent. remain stationary, 25 per cent. die. In this particular case the amount of albumen makes the prognosis unfavourable.

The history is as follows:—Two years before admission he got wet through driving from West Lavington to Salisbury; a fortnight after this he had apparently an attack of acute nephritis, for "water came out all over his body," and his urine "was the colour of tea." The induration of the skin followed closely upon this attack. He states that he has had a little sore place on each leg for over a year. He was formerly in the army, and was much exposed when under canvas in the Zulu war.

CASE 2. *Cervical pachymeningitis*.—Rapid cure. John H—, æt. 50, labourer in gasworks, came to the hospital on November 11th, 1896, complaining of weakness in the hands.

He dates his illness from July, 1896, when he had pain at the back of his neck, so that it pained him to turn his head, and also numbness in his feet, so that he could hardly walk, and felt as if he were walking on pins and needles. In August a copious tertiary syphilitic eruption broke out below his knees. Then his right hand dropped. He had very little pain in his hands.

He was admitted into the infirmary, and his condition was found to be as follows:

Muscles of hands, arms, deltoids, and pectorals all much wasted, exactly resembling the condition found in progressive muscular atrophy. He can close his left hand but not his right. He can flex both wrist-joints. He cannot raise his arms above the horizontal. Measurements:

Upper arm	...	R.	8½	...	L.	8½
Forearm	...	R.	9	...	L.	9
Dynamometer		R.	nil	...	L.	15

Sensation to prick of pin and to heat and cold appears normal, but he several times mentioned numbness as a concomitant symptom.

Well-marked reaction of degeneration found on several occasions in the hand and arm muscles (chosen at random). Mr. Luckham pointed out

to me that very marked changes of nutrition were present in the fingers and toes, the skin of which was quite white and glossy, and a profuse perspiration was often found on the toes. Indeed, the condition exactly resembled that often seen in rheumatoid arthritis. In the fingers the whiteness of the skin ended abruptly at the first interphalangeal joint. This change of nutrition has been observed before.

Charcot mentions that bullæ may occur, and D. W. W. Ord mentioned to me a case under the care of his father where there were marked nutritional changes with formation of bullæ.

The treatment consisted of inunction of mercury ointment, and the administration of iodide of potassium, with hot baths and electricity. A rapid improvement was soon apparent.

November 27th measurements:

Upper arm ...	R.	8½	...	L.	9
Forearm ...	R.	9	...	L.	9½
Dynamometer	R.	17	...	L.	30

December 6th.

Upper arm ...	R.	9	...	L.	9½
Forearm ...	R.	9½	...	L.	9½
Dynamometer	R.	40.	...	L.	50

The patient has now ceased attending, being quite cured and back at his work.

CASE 3. *Œdema of the larynx.*—The causes of œdema of the larynx are manifold. In the mortuary of St. Bartholomew's I once saw the remains of a young woman who had died of œdema of the larynx due to nephritis. The next case I saw was ultimately found to be due to cancer of the larynx, the swollen epiglottis preventing any diagnosis during life. I will now bring before you another variety of this condition. As I came down to the infirmary one day last summer I saw a workman staggering up to the door. I supposed him to be drunk, but he was not drunk—he was being suffocated. Passing my finger into his mouth I found the epiglottis enormously swollen, and at once prepared for tracheotomy. I may say parenthetically that however easy tracheotomy for the adult under an anæsthetic may be, it is a very embarrassing proceeding when your patient is struggling violently and resists your efforts to help him. In fact, it was only when unconsciousness approached that I was able to effect my purpose. The patient was much relieved by

the operation, but sank in a couple of days, apparently from extension of the disease to the lungs. His urine contained a trace of albumen. Now the point to which I wish to draw your attention is this, that in the absence of a post-mortem examination the diagnosis was obscure. Which of the many forms of œdema of the larynx had we to deal with? It is possible that we may know the tree by its fruits, or, in other words, that the remarkable series of illnesses which followed may throw some light on that of my patient. I am indebted to Mr. J. E. Gordon for following the matter up.

First the male attendant who had sat up with my patient in the hospital was seized with pneumonia, and himself came into the hospital to die.

Next the attendant's wife, who had nursed him as long as he remained at home, developed pneumonia and recovered.

Thirdly, the wife's half-brother in a few days developed pleuro-pneumonia, and Mr. Gordon had to operate for empyema.

Now before going further I should like, with your permission, to mention another equally interesting series.

On April 23rd last year I saw in a cottage at Nunton a woman suffering from sore throat. Her illness began with shivering; then fluids returned through her nose. When I saw her the laryngoscope revealed a very swollen epiglottis; subsequently Mr. Luckham admitted her to Pembroke Ward for suppuration of the glands of the neck following the sore throat, and he informs me that the husband a few days after my visit had a high temperature, and soon developed erysipelas of an ulcerated leg; then a lodger in the house who had some slight sore also developed erysipelas. Here, then, we have another opportunity of judging the tree by its fruits,—unless, indeed, we choose to consider that these were several distinct and unconnected illnesses. Whatever view we take of the matter we may at least bear them in mind in reading Sir Felix Semon's recently published paper on "The Septic Inflammations of the Throat and Neck," read before the Medico-Chirurgical Society on April 23rd, 1895, in which he put forward the following thesis: "That the various forms of acute septic inflammations of the throat and neck hitherto described under the names of acute œdema of the larynx, œdematous laryngitis, phlegmon of the

pharynx and larynx, and angina Ludovici, are in all probability pathologically identical."

The heading, for example, of his eighth case is as follows: "Acute oedematous inflammation of epiglottis and arytaenoid cartilages (? purulent infiltration); double patchy pneumonia; right-sided pleurisy; recovery."

But though the author records many cases in which oedema of the larynx has been associated with septic pneumonia in the same individual, he does not bring forward cases where oedema of the larynx in one member of a household has been rapidly followed by cases of erysipelas in the same household.

CASE 4.—The next case I have to bring before you suggests the question, is simple acute laryngitis in children ever fatal? The text-books speak of it as very rarely dangerous if promptly treated by tartar emetic. At 2 p.m. on Jubilee Day I was summoned to a cottage to see a little boy, æt. 4½ years, who had been seized with "croup." In spite of a bath, which the parish nurse had just administered, he was cyanosed and rapidly becoming asphyxiated. There being no time to send for his medical attendant I did tracheotomy then and there on the kitchen table, the boy being too far unconscious to resent the knife, and had him sent to the infirmary. There was no membrane on the fauces, and the child had been running about the house playing that morning, having only had a croupy cough since the day before. The age of the boy and the mode of invasion and decline negative the idea of laryngismus stridulus, although there is no doubt of rickets being present; and I think we must suppose that it was a case of catarrhal laryngitis complicated by the rickety diathesis. Recovery was uninterrupted. An unsuccessful attempt to remove the tube was made on the third day, but we were able to dispense with it altogether at the end of a week; and I think I may say that the house surgeon and I are agreed that in a surgical examination we should refer to his studies any candidate who did not include a penny trumpet in his list of instruments necessary for tracheotomy.

CASE V.—In conclusion, I should like to mention the case of a little boy whom I was asked to see in consultation in the country at a farmhouse. Several members of the family had been ill; the father started with a quinsy, then one of the

children had a sore throat and the rash of scarlatina, and then my patient had a sore throat without a rash. The present difficulty was that the boy could not swallow fluids without their going the wrong way and causing a fit of coughing, nor could he lie down to sleep at night. The mother had had no sleep for two nights on account of his condition. It was feared that the sore throat had been of a diphtheritic nature, and that paralysis of the throat had supervened. On passing the finger to the back of the throat we found a swelling which might in itself be sufficient to cause the symptoms, and judging it to be a post-pharyngeal abscess we were enabled to relieve the condition with very little suffering to the child.

ANTISEPSIS AND ASEPSIS IN OPHTHALMIC SURGERY.

UNDER the above title, Dr. Ludwig Bach, in a special monograph, discusses the subject from the standpoints of clinical experience and bacteriological researches.

Mercuric chloride (1-3000 and 1-5000), aqua chlorini, iodine trichloride, mercury cyanide (HgCy 1.0, NaCl 10.0, aq. destil. 1500), mercury oxycyanide, mercury biniodide, especially for disinfecting the margins of the lids, are the most popular antiseptics.

Recently, formol has been highly recommended; it and the mercury oxycyanide are of lesser effect in coagulating albumen—an important advantage, and also less irritating, while equal in power to sublimate as a disinfectant.

In the pre-antiseptic period suppuration occurred in from 5 to 10 per cent. of cataract operations; at present the losses from infection have been reduced to 1 per cent., and even less. Nevertheless with some operators the former high percentages still occur, although perhaps not always reported.

The method of applying the antiseptic *régime* differs widely, especially in ophthalmic surgery. This is true not only of the agents employed but also of the manner of employing them. Some begin flushing or mopping the conjunctiva and

margins of the lids with antiseptic fluids several days before operation; others apply an antiseptic bandage the day before; the majority defer disinfection of the parts until immediately before operation. How far useless and irrational precautionary measures may be carried is evident from a report published only four years ago; the writer included even the intestinal canal in the antiseptic programme. Five days preceding operation he exhibited calomel followed by salol, magnesia salicylate, and finally, sodium bicarbonate. Not satisfied with syringing the tear-passages only, the nasal chambers and Eustachian tube were treated similarly; the nose was provided with an antiseptic bandage the night before operation.

Latterly, the value of all measures bearing upon this subject has been examined experimentally by means of bacteriological investigation, and much light has been thrown upon it. It has been clearly shown that it is impossible to certainly sterilise the conjunctival sac, much less the margins of the lids, by any known chemical or mechanical means. It has also been proved that a bandage, even an antiseptic one, increases the number of germs by inhibiting winking. It has furthermore been established that gentle mechanical cleansing of the conjunctiva, and somewhat more active rubbing of the margins of the lids, with simultaneous irrigation, is far more effective in diminishing the number of germs than simple flushing.

It has not yet been determined certainly whether the specific antiseptics are more effective in these irrigations than any simple fluid. In some control experiments Bach obtained results but very slightly better when an antiseptic was employed. Others prefer antiseptics. As the result of personal experience with different antiseptics, as well as with the different methods of employing them, Bach sums up his conclusions as follows: Do not trouble the eye too much before operation; avoid all irritation; be accurately aseptic.

In the Würzburg clinic the following preparatory treatment is practised at present. This is not begun until immediately before operation. The lids and adjacent parts are first cleansed with lukewarm water and soap; the margins of the lids are next mopped with a moist sterile pledget of cotton, during simultaneous irrigation with warm normal salt solution (boric acid lotion may be

employed instead). The patient is then directed to wink repeatedly; this promptly floats any germs which may be suspended in the conjunctival sac toward the margins of the lids, and especially into the nose. After winking has been practised for a short period, the lid margins are once more mechanically cleansed with simultaneous irrigation. The speculum is inserted and fixation forceps applied. The region where the cut is to be made is now carefully wiped with a pledget of cotton, during simultaneous irrigation with normal salt solution; the fluid accumulated in the conjunctival sac is mopped up with sterile cotton.

The instruments are carefully sterilised. The following procedures are recommended. Boiling in a weak solution of soda; wiping the cutting instruments with cotton soaked in the following solution: Absolute alcohol and ether, equal parts, with a few drops of ammonia water, then placed for five or ten minutes in a 5 per cent. solution of carbolic acid, and preserved (for not too long a period) until used in sterilised lukewarm normal salt solution; or a 1 per cent. solution of mercury cyanide, as a bath for the instruments, for about ten minutes (Chibret); or a solution of solveol 6.0, lysol 0.1, water 1000 (Burchardt). Every instrument brought into direct contact with the wound, or introduced into the anterior chamber, must be sterilised; the part of the instrument which touches the wound must, therefore, not come in contact with the hand, the lid margin, the fluid in the conjunctival sac—in short, with nothing that is not certainly sterile. If we proceed in this manner, if we cultivate an aseptic technique, the results will be most satisfactory in every respect—rapid healing, safety from infection.

In the Würzburg clinic operation is unhesitatingly practised upon cases with moderate conjunctival catarrh, or with disease of the lachrymal passages, provided a very small amount of mucous secretion is alone present. Nevertheless, proceeding according to the method detailed the results are unsurpassed; the average duration of healing after extraction is ten days. Of 112 extractions during the past year not one was lost from suppuration.

Based upon experiment and clinical experience, Bach would only return to antiseptics and elaborate preparatory treatment if the conjunctival sac and margins of the lids could certainly be made sterile

thereby—a condition which, for anatomical and other reasons, seems impossible. So long as this is the case he would reject every known antiseptic, for the reason that in the concentration and time required as an efficient disinfectant, they are all irritating, injurious, and hence not favorable to healing.

The common argument that bacteria, which may have remained, are weakened in virulence by the addition of antiseptics to the irrigating fluid, and the danger of infection therefore lessened, has been shown by experiments to be fallacious.

"In spite of careful antiseptics" is the apology frequently heard for a bad result from suppuration. What is the use of careful antiseptics, if, for example, an instrument brought into direct contact with the wound is permitted to touch the margin of the lid, the cilia or the fluid in the conjunctival sac? This increases the danger of infection to a far greater degree than where, for example, antiseptic irrigations have been omitted.

Based upon experiment, bacteriological researches, and clinical experience, the author insists that if infection does not take place during operation, it is hardly to be feared that it will occur subsequently from the conjunctival sac; this is, of course, possible, but very little likely if the patient is prevented from pressing germs into the wound by rubbing his eyes, or other hurtful procedure. For infection to occur it is not enough that bacteria should pass over or temporarily lie upon it; they must get into the wound, nay, they must even be pressed in between its lips, as experiment teaches. If this were not so it would be inexplicable that infection does not occur much oftener; in at least 10 to 15 per cent. pathogenic germs are present upon the lid margins and in the conjunctival sac, and remain there in spite of all our endeavours.

What powers, on the other hand, are we justly entitled to ascribe to antiseptic measures? To what extent are we able to influence, by antiseptics, infected wounds, corneal ulcers—in short, cases where infection has already taken place? Schimmelbusch, among others, has instituted experiments which answer these questions. And this is the answer: After infection has once taken place antiseptics are powerless, no matter how employed. As bandages and applications to the eyes were found to have little effect in diminishing the

number of germs upon the margins of the lids and in the conjunctival sac the former may even cause an increase.

Subconjunctival injections of sublimate have been recommended of late years, in some quarters, in ocular affections due to infection of syphilis. Testimony as to their value has been extraordinarily diverse. Chemical examination shows that none, or only the smallest quantity of the medicament, reaches the interior of the eye. Intra-ocular injections dare not be employed.

The case is different with antiseptic ointments. Contrary to the opinion usually entertained that these have none, or only the slightest effect upon germs contained in a liquid menstruum, Bach has found that sublimate and silver nitrate in refined petrolatum are powerfully disinfectant; ointment of yellow precipitate is less effective; boric acid and iodoform are practically negative. These effects hold also in combination with a fluid menstruum, as the tears. Refined petrolatum is the best menstruum.

The improved results of the antiseptic period are to be ascribed (independent of the great importance of antiseptics in the disinfection of instruments) less to the action of these substances upon the germs of the conjunctival sac and lid margins, and upon bacteria in the tissues, than to the mechanical effect of irrigations, superior cleanliness, and better acquaintance with the possibilities and dangers of infection. As practical applications they go hand in hand with increased knowledge gained in the field of bacteriology. As in general surgery and gynecology, so in ophthalmology, the transition from antiseptics to asepsis is being slowly accomplished and will be maintained, unless our armamentarium of antiseptics be enriched by new discoveries which shall surpass those at present known, in many respects. —T. B. SCHNEIDEMAN, M.D., *Philadelphia Polyclinic*, July 3rd, 1897.

Aristol Ointment. — Paucier says that the clumpy product generally obtained by trituration with vaseline may be avoided and an intimate mixture produced by first triturating the aristol with vaseline oil.

New York Medical Journal, Aug. 7th, 1897.

THE CLINICAL JOURNAL.

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CLINICAL LECTURES ON URINE.

Delivered at University College Hospital by

J. ROSE BRADFORD, M.D., F.R.S., F.R.C.P.,

Physician to University College Hospital.

No. II.

Albuminuria.—By the term albuminuria is meant the presence of proteid matter in the urine, but not necessarily the presence of albumin. The first question that presents itself is whether proteid matter is present in the normal urine,—whether there is such a thing as physiological albuminuria. That depends largely on the definition of a normal urine, but apparently 30 per cent. at the very lowest percentage, of apparently healthy people, have some variety of albumen in the urine. The amount present is usually small, and is often not recognisable except by special methods of investigation. There are two ways of looking at physiological albuminuria; one that it is a normal occurrence, and the other is that these patients are really on the borderland between health and disease. It has not been clearly established that cases of so-called physiological albuminuria necessarily go on to organic disease of the kidneys, and to permanent albuminuria. The great point is that there are some 30 per cent. of persons who have no marked symptoms of any disease except the presence of this small quantity of albumen in the urine. In some of the cases the patients have traces of albumen in the urine always, but the greater majority have only albumen in the urine under certain circumstances,—as, for instance, after severe exercise pushed to the point of great fatigue. This accounts for one group of cases. A second group consists of those cases where albumen is only present after meals; and a third group is where albumen is found in the urine passed some two to three hours after getting up in the morning, the so-called postural albuminuria. There is another class, a fourth group of slight importance, in which albumen is said to appear in the urine

after bathing. But this last group is of but little interest. The important groups are those in which albumen appears in the urine after meals, after severe exercise, and in the morning. In some cases the albuminuria appears with such remarkable diurnal constancy as to merit the term cyclical albuminuria.

To consider the albumen which appears after meals; there are certain articles of diet that will produce albuminuria in any person, provided only that enough is taken; for instance, raw eggs will produce albuminuria practically in 100 per cent. of persons. Half a dozen raw eggs will produce albuminuria in almost anyone, and when people are ill it is not an uncommon thing for raw eggs to be taken in considerable quantity; it is worth remembering that albuminuria may occasionally be dependent on the diet that is ordered. Ordinary diet is followed by albuminuria in a considerable number of apparently healthy people, and more particularly in young men, or rather boys. In these cases small quantities of albumen are usually to be noticed two to three hours after the heavy meals of the day. In cases of dietetic albuminuria, and in the albuminuria seen after severe exercise, hyaline casts are frequently found in the urine after centrifugalising, and hence the presence of these casts does not necessarily mean that there is any permanent structural disease of the kidney. There is a particular variety of this so-called functional albuminuria spoken of as cyclical albuminuria, and sometimes known also as Pavy's disease, in which you cannot trace that the albuminuria is dependent on diet or exercise. In these cases of cyclical albuminuria the albumen appears in the urine at more or less regular intervals; thus it may be seen either in the morning or in the evening, but not in between times. These cases of cyclical albuminuria are mainly seen in young persons, and more especially in boys, and it is not an uncommon thing to find patients between fifteen and twenty years of age with a considerable quantity of albumen in the urine. Before, however, the diagnosis of dietetic, cyclical, or functional albuminuria is made, you

ought to be very careful to centrifugalise the urine, and to examine the deposits for casts. You must be on the watch for the presence of spermatozoa, as many cases of albuminuria are due to masturbation, and if you detect the presence of spermatozoa in the urine it will give you a clue to the nature of the cause. Therefore you should not make a definite diagnosis until the urine has been centrifugalised and the deposit examined. It is necessary to centrifugalise the urine, because by simply allowing the urine to stand you do not get enough deposit for your examination. Another reason for centrifugalising the urine is that there is a form of granular kidney in which the albuminuria may be remarkably inconstant, and the mistake of diagnosing a trivial condition, such as functional albuminuria, when there is really a very serious state of things present, *e.g.* granular kidney, may be made, and this mistake may often be avoided by finding definite renal elements in the deposit obtained after centrifugalisation of the urine.

The amount of albumen which is present in these cases of functional albuminuria is often so small as not to be detected by the nitric acid test; that test only yields evidence of albumen in about 10 per cent. instead of 30 per cent. of apparently healthy persons. The picric acid test or some other equally delicate reaction must be used. The amount of albumen is very variable, but usually there is only a trace present. Occasionally, however, larger amounts are present, as, for instance, one tenth, and I have seen a case where there was as much as one sixth, and yet no signs of organic disease were present.

We will pass on now to pathological albuminuria. Albuminuria is brought about, strictly speaking, in only one way, and that is if the epithelial structures of the kidney are damaged, and more especially the glomerular epithelium. Damage to the glomerular epithelium may be brought about in a great variety of ways, and consequently, for clinical purposes, it is convenient to classify albuminuria as dependent on several causes, although as a matter of fact albuminuria is probably always due to a change of some kind in the renal epithelium. The first variety of albuminuria recognised is the albuminuria dependent on changes in the composition of the blood,—so-called blood albuminuria. A typical example of this albuminuria is that which occurs in empyema, or in any case in which there

is a large collection of pus in some part of the body. Albumen occurs in the urine in these cases because the pus in the chest, &c., contains a considerable amount of albumoses; these are excreted by the kidney, and in being excreted they damage the epithelium, and therefore not only do the albumoses pass out, but some proteids of the blood-plasma pass out also. That is a typical example of albuminuria from blood-changes. The primary mischief is the alteration of the blood by the presence of large quantities of albumoses, and this affects the kidneys, with the result that there is an excretion both of the albumoses and also of the proteids of the blood-plasma.

The second variety is the albuminuria from passive congestion of the kidneys, as, for instance, in heart disease, and in tumours, &c., pressing on the vena cava. The albuminuria from partial thrombosis of the renal veins is an extreme example of this form of albuminuria, as is also the albuminuria seen in mitral disease. The albuminuria seen in pregnancy is possibly dependent upon the action of a toxic substance on the kidney, and not simply upon the pressure of the uterine tumour on the renal veins. At one time it was usual to describe an albuminuria caused by high tension; but it is probable that this view is erroneous, and that the albuminuria seen in cases of high tension is really dependent upon an accompanying renal action.

The third variety of albuminuria is that arising from primary organic disease of the kidney or of some part of the urinary tract. To recapitulate the varieties I have described, there is, firstly, albuminuria from primary blood-changes, or toxic albuminuria; secondly, albuminuria from venous congestion; and thirdly, albuminuria from primary changes in the kidney or urinary tract. You may add albuminuria from rupture of an abscess or hæmorrhage into some portion of the urinary tract.

The amount of proteid matter that may be lost in the urine is often very considerable when you bear in mind that you may get nearly as much proteid matter per cent. in the urine as in the blood-plasma. There is about 8 per cent. of proteid in the blood-serum, and the urine rarely contains more than 6 per cent., so that there may be almost the same percentage amount of proteid matter in the urine as in the blood, and hence the loss may be very considerable. The maximum loss may be something like forty or fifty grams daily, and forty

or fifty grams, to put it in more homely terms, is about as much proteid matter as there is in from two to two and a half pints of milk. A patient with certain forms of renal disease may lose from his kidneys very nearly as much proteid matter as there is in a full hospital diet of milk, hospital patients on milk diet taking about three pints of milk per diem. It is a very considerable amount to lose, and though you will sometimes see it stated in books that the amount of albumen lost by the kidneys is not of consequence, I am sure, to say the least of it, that that statement is not a correct way of expressing the fact. In the cases where the highest percentage of albumen occurs, where the urine boils solid, the amount of urine is usually small, and hence you do not get a very great loss, notwithstanding the fact that the urine when boiled is solid. When the urine is much increased there is often a greater total loss of albumen, although the percentage amount of albumen may be much less than when the urine boils solid. The maximum loss of proteid is most often seen in cases of waxy kidney, and more especially, perhaps, in a particular variety of chronic Bright's disease, where the quantity of urine is considerably above the normal, and where it contains a considerable percentage of proteid. The maximum loss of proteid is more often seen in these cases of chronic kidney disease than in cases of acute Bright's disease. The gravity of a case of Bright's disease is not measured entirely by the amount of albumen that is lost; in many of the most serious cases of Bright's disease very little albumen may be lost, and it is possible for severe and fatal uræmia to occur without albuminuria.

The most important varieties of proteids met with in the urine are albumoses, globulins, and albumins. Nucleo-albumins are sometimes present in the urine, and sometimes true peptones are said to occur in the urine; but these are more or less curiosities, and are not of any great clinical importance. The proteids we are really concerned with are albumoses, globulins, and albumins. The first point is never to forget that you may have albumoses in the urine without any other proteid matter, and if these albumoses are present they may not be detected in the ordinary routine examination of the urine. Although albumoses may be present alone in the urine, it is more common for a mixture of proteids to be present.

Albumosuria is, strictly speaking, the name given to the condition where albumoses only are present, but sometimes it is applied to cases where these bodies are present along with other proteids. In febrile states albumoses are found frequently in the urine, and in any disease that is associated with the presence of microbes, and more particularly in cases where the microbes present are those connected with suppuration. Empyema is a typical instance for demonstrating the presence of albumoses in the urine; and the same is true of pneumonia, in which, although there is no suppuration, still there is a fibrinous exudation. In these and other similar cases the albumoses damage the kidney to a sufficient extent to allow a small amount of albumen to pass through, and you then have febrile albuminuria, or what the French call transitory nephritis.

Other febrile conditions besides those associated with empyema and pneumonia probably produce the albuminuria associated with them in this way. At the same time the excretion in febrile states of other toxic substances besides albumoses may lead to albuminuria. This form of albumosuria, with or without albuminuria, and associated with some febrile disorder, is mainly of scientific interest; but there is one form which is important, and that is the albumosuria that occasionally occurs in granular kidney, and perhaps in other forms of renal disease. There are occasional cases of granular kidney in which albumosuria is a marked feature, and the patient's urine may contain quite a large amount of albumoses, far more than is seen in what may be called febrile albumosuria. The only renal disease in which albumosuria is at all marked is this form of granular kidney; but I have seen albumosuria considerable in amount in two cases where the condition of the urine suggested the existence of chronic diffuse nephritis. Albumoses are not coagulated by heat, and if the urine is examined in the ordinary routine fashion their presence may not be detected. It is difficult to understand the cause of this copious albumosuria in chronic renal disease, inasmuch as it occurs without there being any inflammatory complications to cause it. It has been suggested that it has an intestinal origin, and that owing to some disturbance of digestion and assimilation the albumoses formed in digestion are absorbed as such into the blood-

stream, and subsequently excreted. This view is plausible, but there are no facts, as far as I know, to really substantiate it. Another condition in which considerable albumosuria may be present is in ovarian disease. There was a case here recently where the urine contained such an abundant quantity of albumoses as to become nearly solid when the albumoses were precipitated. The explanation of these cases is, as far as I know, unknown, inasmuch as the ovarian tumour was not a suppurating one.

In cases of so-called albuminuria the proteid matter present is usually a mixture of serum-albumin, serum-globulin, and traces of albumose. In many cases of albumosuria there is a certain amount of albuminuria, but the converse is not true. Some years ago an attempt was made to show that the presence of globulin had a different significance from the presence of albumen, but there is no ground for that belief. Peptonuria is said to exist, but many cases are really cases of albumosuria, and cases showing the presence of true peptone in the urine are rare; suckling women are said to exhibit this. Besides confusing albumosuria with peptonuria, there is another possible fallacy; since the urine contains traces of pepsin, if the examination is delayed it is quite likely that peptone may be formed afterwards.

As regards the tests from a clinical point of view, many tests that are suitable in the laboratory are not suitable clinically. Picric acid is a test that is largely used for proteids, and it is a very delicate test, but it has this drawback, that for many clinical purposes it is too delicate; you will get a reaction in these cases of physiological or functional albuminuria, and you may be misled into thinking that it is a serious condition; therefore picric acid is most valuable when functional albuminuria is suspected. It is not a good test to examine urine straight away with. In the country you can carry it solid, which is perhaps a good point in its favour. As regards the ordinary tests, one relies on nitric acid, but different people will give you different advice. Personally I always teach that the nitric acid test on the whole is the best test, and at any rate it ought always to be used in the routine examination of the urine, since by its use albumoses may be detected as well as the ordinary proteids. It is not, however, a very delicate test, it does not

compare in this respect with the picric acid test; but if the urine does not react to the nitric acid test, there cannot be any very serious condition, except some cases of granular kidney, where occasionally only the merest trace of albumen is present, or even none. The nitric acid must not contain nitrous acid, otherwise the urine effervesces, and the essence of the test is that the urine and the acid must not mix. You must float your urine on the top of the acid, and not mix the urine with the nitric acid; if you mix it up the nitric acid may easily dissolve a small quantity of albumen. Another point is that you must leave it to stand for some minutes; if there is only a small quantity of albumen the ring does not come out strongly for some minutes. If you follow the rule of not using nitrous acid, and of floating the urine on the top of the nitric acid, you will have no reason to complain of the test.

The only fallacies are, if there is a small amount of albumen, you may dissolve it with the nitric acid. If you are very careless you may mistake nitrate of urea for a ring of proteids, but that would be a gross mistake to make. The administration of some resinous bodies, *e.g.* copaiba and turpentine, causes the excretion in the urine of a substance that may yield a cloudiness on the addition of nitric acid.

As regards the heat test, you can acidify the urine first, or you can acidify it afterwards. The usual thing to do is to acidify it first. When you acidify, you must be careful to acidify with a weak acid, such as acetic. It is well to boil urine in the usual clinical way, at the top first of all, so as to be able to compare any cloudiness with the lower part of the tube, to see if the opacity increases. If you have a urine that is only very slightly acid or alkaline, and you boil it, the earthy phosphates are precipitated for reasons which we shall see later on; the precipitate in some way resembles a proteid. You clear the point up at once with a drop of acetic acid, but the earthy phosphate fallacy has only to be mentioned to be dismissed. If the urine is not acid enough, and there is only a small amount of albumen present you may have it converted into an alkali albumen, and it will not then be precipitated. The test for albumoses is to take about half a test-tube of urine and drop nitric acid into it gradually. This precipitate of albumose will disappear on heating, but inasmuch

as in many cases of albumosuria there are other proteids also present, when you warm it it may not entirely disappear, the cloudiness may only diminish, to increase again on cooling. That is a rough test for albumoses; and if with this test a result is obtained, the more delicate sulphate of copper and caustic potash test, which gives a violet colour with the proteid matter, and a rose colour with albumoses, may be used as a further confirmatory test. For accurate purposes the albumoses may be separated from other proteids by depending upon the fact that whereas ammonium sulphate precipitates all proteids except true peptones, magnesium sulphate precipitates globulins, and sodium-magnesium sulphate precipitates also albumins; so that by using these salts and washing the precipitates with saturated solutions of the corresponding salts, the albumoses, globulins, and albumins present in the urine may be differentiated. These, however, are methods more adapted to the laboratory, and are as yet of no great clinical importance.

If the urine contains a large quantity of proteid matter, on pouring it into distilled water a precipitate will be seen, and this is dependent on the precipitation of globulin. In an ordinary case of Bright's disease, if you pour the urine drop by drop into distilled water the globulin will be precipitated. In some cases ordinary water may be used where the amount of proteid present is very large. If you are without apparatus of any kind the urine may be acidified with vinegar, and boiled in a spoon over a candle.

As regards the estimation of the quantity of proteid present, the best way is to precipitate the proteid by adding 5 c.c. of the urine to some 50 c.c. of boiling absolute alcohol, and filter; wash the precipitate with alcohol, ether, and water, and then after drying weigh the precipitate; all other methods are more or less inaccurate. It is not generally desirable to determine the amount actually, it is only of scientific interest. The clinical method of determining roughly the amount by the subsidence method is probably as good as any. The urine is boiled after acidification, and the relative bulk of the precipitate to the volume of the urine determined after allowing the urine to settle for twenty-four hours. The state in which the precipitate is thrown down, whether it is a coarse or a fine precipitate, depends on the amount of salts and acids present, and they also

influence the degree to which the coagulum contracts afterwards. Personally, I think the ordinary subsidence method is accurate enough for most purposes. Esbach's method is a modified subsidence method, and is said to be more accurate; it is a question of precipitating urine with acetic acid and picric acid. My own experience of this method in this hospital has not impressed me with its accuracy, and very often the determinations as checked by the weighing method were 100 per cent. wrong, so that personally my advice is to be satisfied with the ordinary subsidence method, unless you wish actually to determine by weight the amount of proteid present.

Now as to the significance of albumen, that is to say, the prognosis of albuminuria. The significance of course varies from a trifling significance in some functional cases to a very grave one. What do you express your opinion upon? You do not express it solely on the percentage amount present: for instance, a pregnant woman may have her urine showing a third albumen, and clearing up completely at delivery, with no permanent ill effects; and some authorities hold that you may have the urine solid with albumen without any permanent structural disease of the kidneys. You may certainly have large quantities of albumen present in the urine, as in pregnancy, without there being any condition which destroys the patient. On the other hand, in a patient with granular kidney you may have little or none, and yet the disease is rapidly fatal, therefore you must not express any opinion on the percentage amount of albumen. In these cases of pregnant women passing large quantities of albumen they pass a very small amount of urine. A patient with a granular kidney passes very considerable quantities of urine, and the amount of albumen is of course more than you would think, hence all considerations of percentage amounts of albumen must always be checked by considering the total quantity of urine. Although of course, broadly speaking, the greater the amount of albumen the greater the damage, this is not always true.

With regard to albumosuria, it is a trifling matter excepting in relation to the granular kidney of which I have spoken, which is always a very severe and grave malady, so the variety of proteid matter is of some but not of great importance. Febrile albuminuria does not leave any permanent lesion

of the kidney. You base your opinion then most of all on the general condition of the patient, the state of the pulse, the presence or absence of dropsy, &c., on the microscopic examination of the urine, and on the nature of the formed elements you may find in the urine, and more particularly what casts you find. I would warn you at once that hyaline casts are not of any great consequence, they are probably nothing but coagulations of the blood-plasma in the renal tubules, and are not of any grave significance; but epithelial and still more fatty casts are of grave significance, so also are waxy casts. The presence of pus and so on will often throw light on the matter, and I want to digress here for a moment. Supposing a urine to contain pus, you must always examine the supernatant portion of the urine, after allowing the deposit of pus to settle. You should syphon off the urine, not stir it up at all, and examine also the upper part. There was a certain patient with a large quantity of pus in the urine, stone was diagnosed, the urine was simply boiled, albumen was found, and there was pus. A nephrotomy was done, and no stone found, and the patient died almost immediately of uræmia; she had pyelitis and amyloid disease of the kidneys. If the supernatant portion of the urine had been examined more albumen would have been found in the urine than could have been accounted for by the pus, and thus the existence of renal disease might have been suspected, and therefore the operation contra-indicated. If you have a large quantity of pus in the urine there must be albumen in the urine from the pus, therefore you let the urine stand and pour off the supernatant fluid; but if you find a quarter or a third albumen in the supernatant fluid, it would be probable that the pus alone could not cause it. In the prognosis of albuminuria you must take into account the state of the circulatory system, the state of the pulse, and the presence or absence of albuminuric retinitis. There is one other remark about the presence of albumen in the urine; the presence of large quantities of albumen interferes with the test for sugar, Trommer's or Fehling's test. If you have a large amount of albumen, a third, quarter, or a half, the sugar reaction does not come off, therefore in examining the urine for sugar, examine first for albumen. If you find a considerable quantity of albumen, precipitate it and filter it off, and then test for sugar. It does

not apply to most cases of diabetes, because in diabetes, even if albumen is present, the quantity of sugar present is usually so large that the test is not interfered with; but sometimes it is otherwise, and the presence of sugar may be overlooked.

REMARKS

ON

THE TREATMENT OF NÆVI.*

BY

HERBERT F. WATERHOUSE,
M.D., C.M. Edin., F.R.C.S. Eng.,

Senior Assistant Surgeon and Lecturer on Anatomy,
Charing Cross Hospital; Surgeon to Out-patients,
Victoria Hospital for Children.

MR. PRESIDENT AND GENTLEMEN,—When I was requested by my friend Mr. A. H. Walker, your Hon. Secretary, to read a paper on some subject connected with surgery before you this afternoon, I thought it might be well if I took as my text the "Treatment of Nævi." My excuse for venturing to take up your time with a few brief remarks upon such a commonplace subject must be that as I have for some time past been accustomed to see in my out-patient clinics between 200 and 250 nævi per annum I have had considerable experience in dealing with these growths, and the results of this experience may be more acceptable to you, who must frequently be called upon to treat such cases, than if I were to have directed your attention to some rare condition which may be met with perhaps once in a lifetime. With this word of apology I will, without further preface, proceed to the consideration of my subject.

I have limited myself to the consideration of the treatment of nævi, and am therefore absolved from the necessity of entering into the difficult and obscure questions of the ætiology and classification of these growths.

The congenital vascular tumour or nævus is for practical purposes best classified according to its location into (1) cutaneous, (2) subcutaneous,

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and (3) mixed. The subcutaneous and mixed are practically almost identical, and demand the same treatment. The cutaneous differ widely from the other two groups, and are far less satisfactory to deal with by operative treatment. At the very outset the question arises, Should nævi be dealt with by operative measures, or should they be left alone by the surgeon? This is a question by no means easy of answer. As so frequently happens, there are two sides to the question, and the *pros* and *cons.* are by no means so unevenly balanced as many would lead us to believe. The more we consider the clinical course of nævi, which have been for some reason or other left alone, the more difficult do we find it to prognosticate regarding the future of any nævus. Nævi to all appearance quite similar may (1) disappear spontaneously, (2) may remain stationary for half a lifetime, or (3) may increase in size with extreme rapidity. It is worthy of notice that those nævi which grow to a large size almost invariably do so within the first few weeks or months of extra-uterine life.

After the first year of life a nævus increases in size only as a rule in proportion to the increase in size of the child. Very frequently the child grows more quickly than does the nævus. The reverse of this is decidedly unusual. In very many nævi that I have watched for four or five years no perceptible increase in size has occurred. We must ever bear in mind that there is a very marked tendency in every nævus to spontaneous disappearance. This I have observed in numberless cases, and I verily believe that it would happen in the great majority of nævi, which do not increase markedly in size, before the period of adult life is reached. This spontaneous disappearance of nævi is of course far more common in the subcutaneous and mixed varieties. In the purely cutaneous forms it is much less frequently met with. I am sure that you will bear me out in the statement that subcutaneous nævi are very rarely met with in persons over thirty years of age. It may be objected that the reason for this is that the surgeon of the present day is not in the habit of letting nævi pass without operating upon them. I freely admit that this accounts in part for the rarity of nævi among adults, but I maintain that anyone who takes the trouble, as I have done, to question closely on the subject will very soon come to the conclusion that the number of nævi which can be shown

to have disappeared spontaneously is far larger than would have been imagined from the surgical teaching of to-day. I would impress forcibly upon you my conviction of the innate tendency present in almost every nævus to spontaneous disappearance. This is, I am convinced, a powerful factor insufficiently recognised in the success attending the treatment of nævi by many various methods. Frequently we meet with nævi which apparently have this tendency developed to such an extreme degree that they require only a slight stimulus to set in motion the changes necessary for spontaneous absorption. Let me mention such a case. A child three years old was brought to me with a subcutaneous nævus, the size of a hen's egg, just below the inferior angle of the right scapula. Into the centre of this the point of a galvano-cautery was inserted at a dull cherry-red heat, and was moved about radially in the midst of the tumour through the same skin puncture for perhaps one minute. The child, a delicate little mite with adenoids, took the anæsthetic so badly, and became so faint, that it was deemed advisable to desist from further destruction of the nævus at the time. A collodion scab was therefore applied, and the parents were told that further operative treatment would be carried out in three months' time. At the end of three months the tumour had hardened and shrunk to a fourth of its original bulk, and six months later, no further treatment having been carried out, the nævus had disappeared, leaving behind it merely a slight fibrous induration the size of a pea. Such cases as these are by no means exceptional. One frequently sees one application of electrolysis suffice to cure a large nævus, though of course four, five, or six applications are usually required. In certain cases the surgeon can with some degree of certainty foretell the progress of the nævus. If the nævus is uniformly compressible, soft, and highly vascular, approaching a bright red colour, especially at the margins, it is fairly safe to predict that it will increase in size. If, however, the nævus be of markedly unequal consistency, thus whilst parts are quite soft and compressible others are indurated; if in addition islands of white skin gradually make their appearance on the discoloured surface and steadily increase in size, such a nævus is probably in process of involution, and will in great measure, if not entirely, disappear. When a nævus

undergoes a fibrous change, this starts generally in the centre of the growth, and proceeds centrifugally. The knowledge of this fact has taught me, when treating nævi by ignipuncture or electrolysis, to endeavour to procure destruction of the centre of the growth as the first step; and increasing experience has shown me that this is a wise measure. In certain cases the process of involution of the nævus is such that fibro-cystic changes result from parts of the mass becoming shut off from the circulation, and degenerating into cysts which are usually multiple, and are embedded in the fibroid tissue of the degenerated nævus.

One of the reasons frequently advanced in favour of the operative treatment of nævi is that there is, as the result of injury, inflammation or ulceration, a great fear of dangerous or even fatal hæmorrhage. This fear is almost entirely groundless; such hæmorrhage, as a matter of fact, does scarcely ever occur. I have very frequently seen cases in which the nævus has, as the result of traumatism, become inflamed and even ulcerated, and I have almost invariably found that as the result of these inflammatory processes the tissue of the nævus grows firmer and hardens, the vessels become blocked with coagulated blood and more or less occluded, and hence hæmorrhage is little liable to occur, and the process of cure is started and often progresses to some considerable extent, or even to an entirely satisfactory conclusion. These accidents have frequently, in cases which I have observed, proved very beneficial. Ignipuncture performed by the surgeon acts in much the same way, but has the advantage that the part operated upon is kept aseptic throughout the process of cure. There can be no doubt that nævi are a cause of much distress to the mothers of our patients; they are blemishes of which the mother naturally enough wishes her child rid, especially when they are situated upon the face and neck, where unsightly disfigurement is caused. I have found it is a matter of difficulty in many cases to induce the mother to allow the nævus to be left alone.

The position I have been, after much thought, induced to take up on the question of the treatment of nævi is that no nævus should, as a rule, be operated upon unless it is distinctly increasing in size, or unless it is situated upon a part where irritation of the growth can hardly fail to occur,

and may be expected sooner or later to give rise to ulceration or hæmorrhage. I include under this head nævi of tongue, lips, palate, rectum. Apart from these somewhat exceptional cases, I maintain strongly that in most cases it is the duty of the surgeon to await events rather than to operate upon a nævus, certainly in a child only a few weeks old. Infants of this tender age bear loss of blood notoriously badly, and during the cure of nævi of some size they are very prone to attacks of grave constitutional disturbance, associated frequently with marked elevation of temperature. When, however, it is clear that a nævus is increasing markedly in size, it is our bounden duty, without delay, to submit it to operative treatment. Cogent reasons for this are that the more the nævus increases in size the more difficulty and risk will attend any operative treatment undertaken for its cure, and it is beyond our power to foretell how extensive a rapidly growing nævus may become. If in doubt whether to operate or leave alone, we must be guided by the size, situation, and character of the tumour.

We may mention ten methods of treatment of nævi:

- (1) External application of collodion.
- (2) Vaccination on site of nævus.
- (3) External application of caustics.
- (4) Ligature.
- (5) Subcutaneous dissection of nævus.
- (6) Injection of coagulants or caustics.
- (7) Multiple scarification.
- (8) Ignipuncture.
- (9) Excision.
- (10) Electrolysis.

Most of these methods have in certain cases their advantages; some of them are, however, never to be recommended. We will take them seriatim.

(1) *External application of collodion.*—This method is often sneered at as a harmless placebo. I have, however, not infrequently seen a nævus disappear under its employment. To what extent the favorable result was due to the collodion I do not venture to say. My own opinion is that the action of the collodion was practically *nil*. This method finds its most useful end in satisfying the maternal mind. If the mother is painting the nævus with collodion, she is satisfied for the time being, and this advantage is sometimes a very real one.

(2) *Vaccination*.—This measure may be unhesitatingly condemned. I have never seen it cure the nævus, and have often observed that the vaccination did not take.

(3) *External application of caustics*.—This method has its use in the cutaneous nævus—the port-wine stain—which is of all nævi the most unsightly and the most unsatisfactory to treat. It cannot be cured without the production of much scarring. If a port-wine stain be situated on any part of the body but the face or neck, I would certainly recommend that it be left untouched. On the face it is, however, frequently so hideously disfiguring, that the patient suffering from it will readily consent to any treatment, however tedious and uncertain, that offers any prospect of real improvement. Unfortunately such nævi differ markedly from the subcutaneous and mixed forms, in that they have little or no tendency towards spontaneous disappearance. The method usually adopted for their relief is that of destruction by the external application of caustics. This of necessity leaves very considerable scarring. Nitric acid, the agent most frequently employed, leaves certainly a more marked scar than does the solution of sodium ethylate introduced by the late Sir B. W. Richardson. This latter is in my opinion the best caustic we possess for cutaneous nævi. Its scars (if the skin, as should always be done, be carefully purified prior to the application of the caustic, and then a collodion and cyanide wool scab be applied so as to prevent sepsis) frequently are not very perceptible, and are certainly less marked than those occasioned by any other caustic. Should suppuration occur the scars are more marked. May I draw attention to the extreme frequency of occurrence of a serious mistake in the treatment of mixed nævi? In these tumours it is necessary to procure destruction of the subcutaneous portion of the growth, and the cure of the cutaneous part as a rule follows. Times without number have I seen cases in which the treatment adopted has been destruction of the skin portion with caustics. This has resulted in ugly scarring, and the subcutaneous portion of the growth has not been in any way influenced for good. This treatment is so essentially erroneous, and so frequently applied, that I venture to enter a strong protest against it.

(4) *Ligature*.—This method I have practically

given up. It has no advantage over excision, than which it is far more painful and much less certain, owing to the fact that parts of the strangulated nævus are very prone to escape obliteration; it leaves an irregular scar, it is apt to cause much malaise and rise of temperature, and sometimes, even in careful hands, starts a local inflammatory process which may travel somewhat extensively. If done at all the operation must be performed with strict antiseptic precautions, but, at the best, it is for nearly all nævi—except, perhaps, some few pendulous forms, such as grow from the lips, vulva, &c.—inferior to several other measures, and is not to be recommended.

(5) *Subcutaneous discission*.—This is, for small and medium-sized subcutaneous or mixed nævi, a really valuable method of treatment; for though in my hands—and I have employed it chiefly for nævi situated on the face—the operation has not infrequently needed to be twice repeated, in every instance I have managed to obtain a cure of the growth without the production of any appreciable scarring. This, of course, for nævi situated on an exposed part of the body is an advantage very highly appreciated by the patient and his or her relatives. The method is thus performed. A cataract needle is introduced about one sixth of an inch from the margin of the nævus through healthy skin, and is passed through the nævus to the opposite pole of the growth. The needle must not penetrate the skin a second time, otherwise hæmorrhage will result, which will necessitate the application of pressure for its arrest. From the original puncture the needle is then to be re-introduced into the nævus a very large number of times, say forty to sixty, each passage of the needle being about one twelfth of an inch removed from its neighbour. All these punctures are made through the same opening. In this way the nævoid tissue becomes lacerated; especially if the needle be well moved about in the substance of the growth, coagulation is started and obliteration of the nævus takes place. This method is simple, efficient, safe, and may be recommended. I have often wondered why it is not more frequently applied. It has yielded me in small subcutaneous and mixed nævi of the face excellent results.

(6) *Injection of coagulants*, as perchloride of iron, and *caustics*, as carbolic acid, may be unhesitatingly condemned. Deaths from embolism and throm-

bosis have occurred by no means infrequently, and in other cases serious sloughing has taken place. Unless the growth is surrounded by a ligature to shut it off from the general circulation, the procedure is dangerous in the extreme, and even when so surrounded, the method one which should in my opinion never be employed.

(7) *Multiple scarification* is, I think, the method which has yielded me the best results of any in those most unsatisfactory cases of cutaneous nævi or port-wine stains. As I have already remarked, such nævi, except when situated upon exposed parts of the body, are best left alone. When, however, a large port-wine stain exists upon the face the disfigurement is so considerable that the patient, as a rule, urges operative treatment upon the surgeon with much persistency, and will not take a refusal. In these cases electrolysis is useless, and excision impracticable. Our choice lies between the destruction of the growth by the external application of caustics and multiple scarification. If the nævus be small I attack the whole at once, making a number of parallel linear incisions, say twelve to the inch, and then a second series at right angles to these. This divides the port-wine stain into little squares of about $\frac{1}{2}$ of an inch in diameter. Should the nævus be extensive I operate upon a portion only of the growth at one sitting. After scarifying the nævus I rub over the surface solution 1 to 10 of carbolic acid in absolute alcohol. From this method I have had in port-wine stains results superior to any I have seen attained in any other way. The application is tedious, uncertain, and so intensely painful as to necessitate the employment of a general anæsthetic, but in spite of these undoubted drawbacks I cordially recommend it. I take it that the scarification severs the blood-vessels, and that the alcoholic solution of carbolic causes an acute aseptic inflammation, which aids in the obliteration of the vessels.

(8) *Ignipuncture*.—By this method is meant destruction of the nævoid tissue by the application of the fine needle-point of the Paquelin cautery, or far better by the galvano-cautery. I have for a long time used this method with marked success. The heated needle, which must be kept at a very dull cherry-red heat, is plunged into the interior of the nævus, preferably in the centre of the discoloured skin of a mixed nævus. Through this

single opening in the skin the needle is moved about in all directions, so as to thoroughly destroy the nævus, and on its removal the opening is covered by a collodion scab, and the slough caused by the cautery is in time absorbed aseptically. Points of importance are that the skin be thoroughly cleansed with an antiseptic before the needle is introduced, and that the latter be kept at a dull cherry-red heat. If hotter than this severe bleeding will occur, whilst at the heat indicated the operation is entirely bloodless. Not infrequently this operation requires to be repeated once or twice, but success is invariably attained in the long run. I employ this method chiefly in cases of mixed nævi. It is important to note that in mixed nævi curing the subcutaneous part of the growth cures the cutaneous portion at the same time. One disadvantage of ignipuncture is that the slough takes a long time to become absorbed, and during the whole of this period the child is apt to suffer from considerable malaise, even though an aseptic condition is maintained throughout. For small stellate nævi, such as are not infrequently found on the face, there is no treatment so effectual and so satisfactory as the puncturing of the centre of the star with the fine point of the galvano-cautery. Ignipuncture is, especially when fine platinum points are employed with the galvano-cautery, a really valuable method of treatment in many forms of nævi.

We have now discussed several methods of treating nævi, and have seen that most of them have their advantages; still the vast majority of subcutaneous and mixed nævi will be treated by one of the two remaining methods—(9) excision, or (10) electrolysis. In discussing the relative merits of these two methods it is essential to bear in mind that whilst excision has the advantage of being rapid, thorough, and easy of performance, it, of necessity, leaves a scar; that electrolysis cures a nævus without scar formation, and that it is applicable to any subcutaneous or mixed nævus, however large and formidable. Many nævi are thus too large and too formidable for excision; none are beyond the range of electrolysis. In brief, we may state that excision should be the method chosen for nævi situated upon parts of the body covered by the clothes, provided that their size is not too great. Electrolysis is to be preferred for nævi situated upon the face and neck, where the

avoidance of a scar is a matter of real importance, and for such nævi as from their size are clearly unsuited for excision.

(9) *Excision*.—This method is, I take it, that most frequently employed for the treatment of nævi. It has many advantages in parts where the presence of a scar is unimportant. Union by first intention can practically always be obtained; nothing is left to slough out or to become absorbed; there is no need of repeating the operation, as at one sitting the whole growth can be rapidly and cleanly removed. In the great majority of these nævi there is a distinct fibrous capsule, which renders the excision of the tumour much easier. It is a mistake, however, to imagine that every subcutaneous nævus is encapsulated, though it cannot be denied that the great majority are. A purely subcutaneous nævus is not frequently met with; most of the growths apparently subcutaneous involve the deeper layers of the skin to a greater or less degree, and in these cases the capsule never extends into the skin to which the tumour is always closely attached. Frequently when reflecting skin in the neighbourhood of a nævus it is necessary to preserve some that is partly nævoid in appearance. When necessary this may safely be done, as such skin almost invariably begins soon to take on a normal appearance. The danger of excision is hæmorrhage. This may be avoided by cutting wide of the nævus, by operating rapidly and with skilled assistants, and by passing under the growth four to six large steel pins, below which a piece of rubber tubing is tightly wound and securely tied. By this means the nævus can be removed almost bloodlessly, its feeding-vessels, which are by no means as large or as numerous as one would imagine, are ligated, and the sutures are introduced, though not tied before the pins and constricting tubing are removed. Such a procedure has many advantages, and is much to be recommended. Excision should be in all cases the method of treatment adopted in cystic degenerated nævi and lipomatous nævi.

(10) *Electrolysis*.—By this method properly and patiently applied any subcutaneous or mixed nævus, however large and formidable-looking, may be safely and certainly cured. This can be said of no other method of treatment. There are undoubtedly, however, disadvantages connected with electrolysis. The apparatus required—battery,

needles, &c.—need a certain amount of care and attention; but the main objection to the method is that in my hands, at least, a nævus of any considerable size often requires four to six operations, each one under a general anæsthetic, before it is entirely cured, and that an interval of six to eight weeks must elapse between the various operations. These objections suffice to explain the comparative infrequency of its employment among hospital patients. Many surgeons imagine that in order to perform electrolysis satisfactorily it is necessary to have an intimate acquaintance with electricity, and to use a galvanometer and other instruments. I am sorry to say that my electrical knowledge is practically *nil*, but I have, nevertheless, had excellent results in the electrolysis of nævi. I have never employed a galvanometer. A current of fifteen to twenty milliampères, which can be got from a simple arrangement of any cells, is all that I have found necessary for even large nævi. I have always worked on the principle that success in electrolysis depends upon not doing too much at one time and in one place, and I have therefore but very rarely had any sloughing, notwithstanding the fact that the action of electrolysis is purely destructive, and that it cures a nævus only by destroying and obliterating the vessels which make up the substance of the growth. I take it that the caustic potash and nitric acid produced in such minute quantities by the electrolysis of the tissues, by destroying the tissues in their neighbourhood, occasions the production of minute sloughs, which start the process of cure by fibrous metamorphosis, much in the same way as a similar process is started by ignipuncture, though the latter is a far coarser, less delicate method. The needles that are used should be those introduced by Prof. Fraser and Mr. J. Duncan of Edinburgh, the latter of whom is certainly the highest living authority on the subject of electrolysis of nævi. They should be insulated to within one third or half an inch of their points with vulcanite. Steel is certainly the most satisfactory metal, but the positive needle, owing to its being acted upon by the electrolytic current, becomes corroded. I generally employ common sewing needles, roughly insulated with vulcanite to within one third to two thirds of an inch from the point, and throw away the positive needle each time after use. In large nævi it is necessary to introduce the needles

through several openings. It is necessary to observe accurately the extent of the induration that soon surrounds each needle. I generally keep the needles in the substance of the vascular growth until they become surrounded by pale doughy masses, which begin to crackle somewhat from the gases given off around them. If one waits for a greater amount of induration, and until these parts become quite white, there is a considerable risk of sloughing. Into the subject of electrolysis and its results I would that time would allow me to go further, but I fear I have already reached my time limit, and must trespass no longer on your kind attention. I would add that not only *nævi*, but almost all kinds of vascular tumours, especially cirroid aneurysms, are generally best treated by electrolysis. I have as yet to meet the subcutaneous or mixed *nævus*, which cannot be cured by electrolysis, though I desire distinctly to emphasise the point that I do not recommend the method for every case of *nævus*, excision being greatly preferable for medium-sized *nævi* so situated that a scar is a matter of no importance. In my experience electrolysis is useless for cutaneous *nævi* (port-wine stains), and numerous *nævi* amenable to its influence would be better treated by other methods. Still, it is for many *nævi* an almost perfect method, and for not a few the only safe and admissible curative procedure.

EXCISION OF THE TONGUE BY WHITEHEAD'S METHOD.

BY

ALFRED PARKIN, M.S.,

Senior Surgeon to the Victoria Hospital, Hull.

REMOVAL of the tongue through the mouth probably to an onlooker appears to be a very dangerous operation, but to a skilful surgeon it presents no difficulties which cannot be promptly and efficiently met, and the results at the present day are so satisfactory that it is only on rare occasions that cutaneous incisions have to be made, whilst a preliminary tracheotomy is still less frequently required.

Since the time that Mr. Whitehead first described the operation which goes by his name, several slight modifications have been introduced in the technique only, the result of greater familiarity with the operation; and whilst by some it may be considered that such details are quite an unimportant part of the procedure, the majority of surgeons will be ready to admit that the success of an operation often depends on a careful attention to trivial points learnt only by practice.

The severity of the operation of course depends upon the nature and extent of the growth, the mobility of the tongue, and the amount of extension from the tongue to the floor of the mouth, fauces, or even to the jaw. It is not necessary here to discuss when a mouth operation alone would be insufficient, this being a point depending on the ability of the surgeon as well as upon the extent of the disease.

Some surgeons advocate the entire removal of the tongue when only one half of that organ is affected, and the disease well limited; this is a point which can be best settled by carefully made statistics, but the advantage to the patient of retaining half the tongue is considerable, and the risk in removing the whole is greater than in removing the half certainly by the method described below.

Without stopping to recapitulate the details of preparation of the patient, position, or other preliminary steps, which vary for each operator, it will be found that the tongue will be best controlled by a stout ligature passed through the affected side and held by the surgeon. Whether the half or the whole tongue is to be removed it is certainly advantageous to split the tongue vertically by cutting down the length of the dorsum with scissors; after division of the mucous membrane the two halves of the organ can be separated easily with the fingers, or if necessary with a blunt dissector; the unaffected side can now be easily held out of the way with tongue forceps or by another silk suture.

Some operators now cut through the mucous membrane of the floor of the mouth, the fauces, and dorsum of the tongue, but it is much simpler to raise the tongue well up with the tip to the nose, and divide the mucous membrane of the floor of the mouth only; and if a sponge be placed in the corner of the mouth, close up against the

anterior pillar of the fauces, it is impossible for any blood to trickle down over the epiglottis.

It is easy to cut through the base of the tongue with scissors boldly and rapidly; at each cut the tongue comes more out of the mouth, and the section in the mouth can be made as deep as required. The upper surface of the tongue being uncut, really acts as a shutter to prevent any blood passing backwards, and the sponge in the outer angle prevents any trickling down that way.

Any spurting is naturally directed by the surgeon straight out of the mouth, generally on to himself, and the vessels can be secured in any way that he prefers with the greatest deliberation. The last part of the tongue to be divided is the dorsum and anterior pillar of the fauces, and this will not usually be done before all bleeding points are secured. If necessary the other half of the organ can be rapidly removed in the same way.

Formerly it was customary to leave the raw surface in the mouth to granulate up as best it could, to the great discomfort of the patient, and prolongation of the period of recovery; but it was suggested by Mr. W. Arbuthnot Lane in the 'Lancet,' 1892, vol. i, p. 1291,* that it was very easy to so cut through the mucous membrane in the early stages of the operation that a large flap remained, which, when only half the tongue was removed, could be easily sewn to the raw median surface of the unremoved part, in some instances completely covering all the raw floor of the mouth. I have invariably followed out this plan with the greatest benefit to the patient, in that there is much less soreness of the mouth, there is less risk of septic infection of the lungs, and in some cases it is possible to obtain healing by primary union along the whole length of the incision, and consequently a remarkably rapid recovery. The mucous membrane of the mouth is so movable when healthy that it is seldom that one cannot by a little careful manipulation, even when the whole tongue is removed, considerably minimise the amount of raw surface left, and this can be done without in any way cutting near the diseased part of the tongue itself. It is scarcely necessary to add that suturing the mucous membrane over the raw surface in this way is a capital aid to the prevention of hæmorrhage after the operation, and

cannot but add to the comfort of the surgeon when such an operation is done away from home, possibly in the absence of any immediate skilled assistance.

The minor details of painting the mouth with iodoform solution or other similar preparations, and the subsequent frequent cleansing of the mouth with antiseptic washes, require no further mention—it is so obvious that the cleaner the mouth is kept the more rapid will healing be. I have, however, found it a great advantage during the first few days to place a piece of gauze in the floor of the mouth, with its end hanging well outside; this readily conducts most of the discharge away after the fashion of a dentist's salivary pump, adds considerably to the comfort of the patient, and prevents any discharge being swallowed or possibly trickling down the trachea. It does not follow that because the mouth is a septic cavity that no care should be taken to treat wounds inside it as aseptically as possible.

Some surgeons might argue that small details of this description are, to say the least, unnecessary; to these one might say that surgery ought to be (it often is anything but) the finest art; and that if I had to be operated on myself I would prefer a man who would, if it were possible, leave me half my tongue, who would prevent any blood whatever from trickling down my throat, and who would adopt all possible means to obtain a satisfactory healing of the resulting wound with the least risk of hæmorrhage or septic trouble, and with the least possible inconvenience to myself during the process.

Lavage of the Blood through the Medium of the Stomach.—Mausel suggests ('Rev. d. Thérapeut.,' May 15th, 1897) that in the discussion about the "lavage of the blood," which can be accomplished by subcutaneous and intravenous injections of artificial serum, the use of the digestive tract as an agent for absorption has been overlooked. Absorption by this route is less rapid, but equally as sure, as by the other, and the subsequent diuretic action is just as efficacious. This is the revival of the administration of "cooling draughts." Preliminary bloodletting is important, as it removes a part of the poisonous material and also favours the absorption of the saline solution into the circulation.—*Med. News*, N.Y., July 31, 1897.

* "A Modification of the Operation of Partial or Complete Excision of the Tongue."

NOTES.

The Treatment of Cancer by the Electrical Diffusion of Nascent Oxychlorides of Mercury and Zinc.

G. BETTON MASSEY, M.D., in the *Charlotte Med. Journ.*, July, 1897.

THE cancerous affections, carcinoma and sarcoma, have been successfully treated by electricity at various times in the past.

Granting the undoubted occurrence of cures by this agent in the many isolated cases reported, it is of interest to inquire why the method has not been more generally recognised, in view of the reported increase of mortality from this affection under the recognised treatment. This reason was most likely of a three-fold nature, including an inadequacy of the method in advanced cases, uncertainty of dosage from lack of acquaintance of the operators with means for measuring the current, and the general dependence of the profession of late on cutting operations.

The revival of the remedy under modern conditions of electrical knowledge is due to Steavenson and Inglis Parsons of London, the latter succeeding in curing a number of mild cases by massive doses of the current, flashed and reversed through the growth from needles of platinum inserted into it and attached to both poles.

It was with these encouraging precedents that I began experimenting with carcinoma of the cervix uteri a number of years ago, and later with sarcoma of other portions of the body, and after several preliminary publications I am now in a position to report results, which, though few in number and not always successful, are yet of truly momentous importance to the human race.

For as a result of these experiments I can confidently announce that a real cure has been found for sarcoma in accessible situations, and a probable cure for carcinoma similarly situated.

The principle of my method is the interstitial diffusion and impregnation of the morbid growth with nascent oxychlorides of lethal metals by electricity in massive doses. The destructive effect of caustics on these growths is well known, but they act only where placed, exactly as the knife does. By my method a relatively infinitesimal

portion of the oxychloride acts lethally on the cancer cells because of its nascent condition, and because it is carried by the current into the very cells themselves. But the most important point is that by the method we may cause the medicinal laden current to seek out and follow the paths of proliferation of the growth, failure to destroy which is the cause of its reappearance after attempts at extirpation with the knife. It is evident that the cancer cell has less physiological resistance to this interstitial attack than normal tissue, for it is found to lose its vitality at some distance from the electrode without causing necrosis of the healthy tissues, though all life in the central portion of the mass will succumb to the method.

The metallic substance is diffused by electrolytic cataphoresis—that is, it is produced by the electrolytic destruction of a metal placed within the substance of the growth, the atoms of the metal uniting with the oxygen and chlorine, and also with the albumen of the tissues, and this compound, together with still nascent atoms of the metal, is conveyed physically into the tissues by what is called cataphoresis, following the lines of least resistance towards the other pole. Considerable diffusion occurs in all directions beneath this active pole, since the lines of current flow spread out into a widely separated brush-like shape; hence for a proper saturation it is necessary to bring the metal in close contact with all portions of the growth, or else to employ a strong current to convey an efficient density to the desired point.

This diffusion of a metal from a corrodible electrode occurs only at the positive pole; it is therefore necessary to connect the active electrode to this pole of the battery. Up to the present time my work has been conducted with active electrodes of zinc, heavily coated with mercury, though it has recently occurred to me that gold and mercury would be a better combination. In action the zinc (or gold) becomes smaller by erosion, but the greatest loss of substance occurs at the mercurial coating, which must be renewed before each use of the electrode. The exact constitution of the metallic oxychloride thus formed has not yet been determined, but it is most probably in the main an oxychloride of mercury.

The cardinal point of the method being a quick and complete saturation of the growth and its ramifications, it is essentially monopolar, the

position of the negative pole being so arranged that the whole of the possibly diseased area shall be traversed by the current on its way from the positive or active pole to the negative. This is accomplished in two ways, as follows :

If the growth be small, the active electrode is placed within it, and the negative, in the shape of a large pad, on some indifferent surface of the body. Under this arrangement the current traverses a large portion of the body, and on account of the diffusion that will occur through healthy parts I have never employed more than 400 milliampères in this way. Cocaine anæsthesia, simultaneously produced by cataphoresis, is sufficient up to 150 milliampères, but the pain developed above this amperage makes general anæsthesia often best. At the lower current strength it is essential that the application be daily or thrice weekly, and kept up until the whole of the morbid area has been either destroyed or changed into normal tissue before the cavity thus made is allowed to heal. Besides small and recent growths this method is also applicable to inoperable growths of large size in which the situation of blood-vessels or important organs renders the more massive and immediate destruction unwise.

The second method, in which an operable sarcoma or carcinoma is destroyed at once, is applied as follows : The patient being anæsthetised, a dozen or more lancet-shaped amalgamated zinc electrodes are inserted around the periphery of the growth, just beyond the area of infiltration, each electrode being attached to one of a leash of fine wires that lead to the positive pole of the battery. The negative pole, a disc covered with absorbent cotton saturated with a solution of potassic arsenite, iodide, or some other salt with a lethal electro-negative radical, is placed in contact with the centre of the growth itself. It will be necessary to make openings in the skin or mucous membrane for the zinc lancets, owing to their brittleness and dulness. Everything being in readiness, a current of at least a thousand milliampères is now turned on through a controller and reliable meter from a current source of proper voltage. The growth will blanch and shrink at once, while areas of destruction appear about each zinc blade and probably in the centre. After a duration of from five to fifteen minutes according to the size of the

growth, the current is turned off, the electrodes are removed, and a dry dressing applied.

Such is a brief outline of the methods and their technical details, but it should be understood that these details are subject to change in adapting the method to individual cases. The underlying principle that constitutes the novelty is that there is a virtue in the electric diffusion of nascent chemicals throughout a malignant growth which, when of sufficient density per area, will cause an interstitial death and ultimate absorption of the malignant cells at a distance from the electrode, without destroying the connective tissue surrounding them, and that this intra-cellular lethal action is independent of and additional to the ordinary destructive action of a strong current in the immediate neighbourhood of the electrodes. With this important fact established the cure of cancer in certain locations becomes a mere engineering problem in this age of electric power.

Pain in the Diagnosis of Cancer of the Breast.—There is still a prevalent belief that cancer of the breast is painful in all its stages. Patients continually express surprise when such cancers have reached an advanced stage without pain. This belief is probably due to the failure to discriminate between the early and late symptoms of the disease. In the late stages, after surrounding structures are pressed upon, or ulceration exists, more or less pain is generally felt ; but in the early stages it is seldom present.

In order to study the relationship between pain and beginning cancer, and to draw attention anew to the fact that tumours should not be considered bland because they are painless, I have examined the histories of the patients who have been treated in the New York Cancer Hospital for mammary cancer since January, 1889. In the history blanks there is a space for recording the presence or absence of pain and any other pertinent facts concerning this symptom, and this has usually been filled in with care.

The total number of cases was 331 ; in most of them the disease was far advanced.

In 63 cases pain was denied in any stage of the disease.

In 56 cases pain was not mentioned.

In 190 cases pain came in the late stages of

the disease after the surrounding structures were pressed upon.

In 20 cases pain was present in the early stages of the disease.

Hence in only 6.04 per cent. of the cases is there a record of pain at the time when the diagnosis should have been made in order to secure the best results from operation. Even in these few cases the pain was of such a character as to give little alarm to the patients.

In 5 instances it followed bruises.

In 1 instance it was caused by what was supposed to be a boil.

In 7 there were only slight shooting or aching pains, which hardly attracted attention at the time.

In 6 there were severe pains at the beginning, which intermitted more or less afterwards.

These 6 cases represented the greatest amount of early pain which was recorded in the entire series of 331 patients. Yet the average duration of their disease had been twenty months when they were admitted to the hospital—an indication that the pain had not been very troublesome, else it would have been relieved sooner.

Even in the later stages of the disease pain was not a prominent symptom. Many of the patients who denied pain at any time had advanced growths; and in the group of 190 patients who had pain late in the disease there was seldom much suffering from this cause. The records usually say: "Occasional shooting pains," or "Dull pain from time to time." I have seen severe pain accompanying cancer of the breast only when there was abscess or phlegmon in addition to the cancer, or when axillary pressure caused oedema of the arm.

The explanation of this absence of pain is to be found in the peculiar nature of the growth. As the cancer grows it replaces the neighbouring normal structures; it does not distend them, as acute exudative inflammation does. Abscess of the breast and acute articular rheumatism illustrate this point, when contrasted with beginning cancer of the breast, tuberculous joint disease, and syphilitic dactylitis. The first are very painful; the last seldom give pain, excepting when motion or trauma brings pressure on nerve-endings which are still sensitive.

Growing fibrous tumours in the breast are more likely to give pain than is growing cancer. There were thirty-seven such treated at the hospital during

the time mentioned, and nineteen are recorded as painful in the early stages, the pain being usually of a sharp shooting character, frequently most severe at or about menstruation. This indicates that if a small nodule is painful, it is more likely to be fibrous than cancerous.—CHARLES N. DOWD, M.D., *Medical Record*, August 7th, 1897.

Ichthyol in Ophthalmology is destined to occupy as important a place as in dermatology and gynecology, this is announced by those who have made a special study of its effects in all kinds of conjunctivitis and blepharitis. Jacovidés in the 'Revue Méd.' of February 3rd describes his experience with it, and states that it affects all the inflamed tissues by vaso-constriction, having also an analgesic effect. It modifies and dries up the conjunctival secretions by its astringent action (except in neonatorum), and in cases of pannus it is invaluable in clearing up the cornea.—*Journ. of Amer. Med. Assoc.*, July 24th, 1897.

Influence of Habitual Inclination of Pelvis on the Pelvic Canal.—Hirst ('University Medical Magazine') points out that although the pelvic obliquity on account of its normal variations can generally be neglected in regard to childbirth, the influence of habitual inclination of the pelvis on the development of the pelvic bones and pelvic canal has been neglected. Exaggerated inclination of the pelvis in childhood, so that the trunk weight is received by the sacrum, increases the forward rotation of that bone, diminishing the antero-posterior diameter of the inlet and the depth of the canal, while excessive action of the rotator muscles of the thigh tends to widen the outlet. On the other hand, if the pelvic inclination is diminished, the top of the sacrum is pushed backward, the antero-posterior diameter of the inlet is increased, as is also the depth of the canal; while pull on the iliopsoas muscles, separating the iliac bones apart, and by compensatory displacement of the ischia, narrows the transverse diameter of the pelvic outlet.

Indian Lancet, July 16th, 1897.

An entirely new work on 'The Diseases of Women,' written for students and practitioners by Mr. Bland Sutton and Dr. Giles, is now in course of issue by the Rebman Publishing Company.

THE CLINICAL JOURNAL.

WEDNESDAY, SEPTEMBER 1, 1897.

CLINICAL LECTURES ON URINE.

Delivered at University College Hospital by
J. ROSE BRADFORD, M.D., F.R.S., F.R.C.P.,
Physician to University College Hospital.

No. III.

WE will consider to-day the question of the colour of the urine, that is to say, the pigments, normal and abnormal, that the urine may contain. The urine is described as being normally straw-coloured; the depth of colour, however, varies considerably. As regards the cause of the normal colour, we must consider mainly the points that are of clinical importance. It used to be taught that the urine owes its colour mainly to a single pigment, urobilin, but that in addition to this coloured body it was stated that the urine contains a colourless substance spoken of as a chromogen, which on the addition of an acid becomes converted into urobilin or a modified form of urobilin known as febrile or pathological urobilin. There is, however, no doubt that this view is not correct, since the pigment causing the normal yellow colour of the urine is not soluble in chloroform, and, on the other hand, urobilin is soluble in chloroform. The colour of the urine is therefore certainly not entirely due to urobilin, but the urine contains some urobilin. The yellow colouring matter to which the urine owes its colour is best called, as Thudichum called it, urochrome. This substance can be extracted by appropriate methods from the urine, and it is found that it does not yield a banded spectrum; and the bulk of the colour is due to this yellow pigment, urochrome. In addition to urochrome there is a certain amount of urobilin, a pigment also found in the bile. Whereas urochrome is insoluble in chloroform and yields no spectrum, urobilin is soluble in chloroform and yields a definite spectrum. In the normal urine there is also a certain amount of uroerythrin, a substance which colours urates pink, and which is present in greatly increased amounts in various

diseases. Another pigment present in the urine in small quantities in health is the iron-free derivative of hæmoglobin, hæmatoporphyrin. There are thus at least four pigments in the urine: urochrome, which is the most abundant, urobilin, which is present in small amounts, uroerythrin, in smaller amounts, and hæmatoporphyrin, in traces only. In addition to these pigments the urine contains a number of substances which are perhaps best described as chromogens, bodies which do not impart a colour to the urine, but which under the influence of oxidising agents become coloured. Some of these chromogens are bodies like indol and skatol and their allies, which are present, combined with sulphuric or glycuronic acid in the form of aromatic sulphates. Thus, indoxyl sulphate of potash and skatoxyl sulphate of potash are substances which are not themselves coloured, but which on the addition of mineral acid yield coloured bodies, and assist to form the ring of colour which is seen on pouring urine on to nitric acid. One of the most interesting of the bodies coming under the classification of substances which are not pigments till they are oxidised, is the body indican (there are 20 mgrms. of this substance in the urine in the twenty-four hours), and this is readily converted into indigo by adding hydrochloric acid. The facts that one wants to impress are that, first of all, there are at least four pigments in the urine, and that besides these, there are other substances which are not pigments, but which become so on the addition of acids. A further point of interest, as regards the origin of the urinary pigments, lies in the fact that if owing to a biliary fistula a patient discharges the whole of the bile through the abdominal wall to the exterior, you will find the urine apparently quite normal in colour. Thus, if no bile enters the intestine, the urine still retains its typical straw colour; that is a fact that has been known for several years, and it is a very serious objection to the view that the urinary pigments are formed from retrogressive changes in the bile pigments in the bowel. A patient I saw discharged the whole of his bile through a fistula, and at the end of a year

his urine was still quite typical in colour. The question of the origin of the urinary pigments scarcely concerns us here, but the blood pigment of the body and of the food in addition to the bile pigments may be the sources from which these urinary pigments are ultimately derived.

Urobilin is readily separated from the urine, and sometimes it is of interest, I cannot say it is of great importance, to do so in certain diseases, pernicious anæmia for instance; all that is necessary is to take some urine and precipitate it with lead salts, the precipitate of lead salts carries down the pigment with it, and this is collected on a filter, and extracted with alcohol containing a few drops of sulphuric acid; in this way the urobilin is obtained in an alcoholic solution, and its spectrum can be readily determined, and this is a much more satisfactory method than that of examining the urine itself spectroscopically. The normal urine rarely yields more than a shading at the violet end of the spectrum; but if a large quantity of urobilin is present, the band typical of this substance may be seen without extracting the urine as described.

In regard to the colour of the urine it undergoes great changes in disease; thus it may be almost colourless, as in diabetes insipidus, greenish as in diabetes mellitus, brownish black as in melanuria, blue as in some cases of indicanuria, greenish-brown in jaundice, red in hæmaturia, black in carboluria, &c. A satisfactory classification of the pigments present in disease is rather difficult of attainment, and I will, therefore, only divide them into two groups, the first consisting of the conditions where the abnormal colour of the urine is dependent upon the presence in excessive amount of some normal urinary pigment, and the second group where the abnormal colour is dependent on the presence of an abnormal pigment. We will consider the group where the abnormal colour of the urine is due to the presence of a normal pigment in excessive amount first.

Urobilinuria.—Urobilin is present normally in small quantities, and does not appreciably colour the urine, the colour being due to urochrome. When there is much urobilin in the urine, it is usually brownish-red in colour, but it may be more brown than red, so that it is mistaken for bile, but on the other hand it may sometimes be sufficiently reddish that it is mistaken for blood. I must warn you at the very outset that urobilinuria is often not

recognised, and many mistakes are made when there is a very large amount of urobilin present in the urine; competent observers may think from the naked-eye appearances that the urine contains blood or bile according as it is red or brownish. Patients who pass a large amount of urobilin in the urine are frequently yellow, that is to say, they are apparently jaundiced, and sometimes they are very yellow, but they are not really jaundiced if you use the word in its strict sense, and that sense is a person who is yellow from the presence of bile pigments in the tissues of the body, and this is not of course synonymous with a yellow person.

The cause of urobilinuria is usually simple, it is due to a decomposition of blood-pigment, a breaking up of hæmoglobin in some part of the body, and this breaking up of hæmoglobin may occur inside the blood-vessels, as in the disease known as paroxysmal hæmoglobinuria, or external to the blood-vessels as in other maladies.

Paroxysmal hæmoglobinuria is usually an affection of people who have lived in hot climates; its nature is not fully understood, but the red blood-corpuscles are more fragile than normal, and the red blood-corpuscles disintegrate, apparently within the vessels, and the hæmoglobin is liberated into the blood stream and excreted in the urine in the form of hæmoglobin, methæmoglobin, and urobilin. Another instance of urobilinuria dependent upon increased blood destruction is afforded by pernicious anæmia; in a marked case of this disease the patient is lemon-tinted and the urine is dark, notwithstanding that the patient is extremely anæmic, whereas most patients with anæmia pass a urine light in colour, these patients with pernicious anæmia are yellow and pass a dark urine. Both the lemon tint of the patient and the dark colour of the urine are due to the presence of urobilin in excess. Another instance of a slighter degree of urobilinuria is seen in septicæmia; many patients with septicæmia or pyæmia are yellowish and pass urine dark from excess of urobilin. One of the most interesting and important forms of urobilinuria is where the condition arises as a result of a large internal hæmorrhage, which is very frequently abdominal. These cases of abdominal hæmorrhage are often due to causes which do not necessarily lead to death, whereas large hæmorrhages in other situations are perhaps more often immediately fatal. A large hæmorrhage in the

pleura, for instance, is usually due to rupture of an aneurysm, and the patient dies rapidly; but not uncommonly you get large hæmorrhages into the peritoneum or retro-peritoneum without death occurring, at any rate, immediately, and it is very striking to watch these cases. A few days after the hæmorrhage the patient may become feverish and apparently jaundiced, and passes urine which is dark brown in colour, and often supposed to be bile-stained, whereas it is really urobilinuria. You will probably see such a case sooner or later, and it is a thing that when once seen you will have no difficulty in again recognising. A case in point is that of a man who was operated on for hernia and had some omentum removed; some hours afterwards he became rather collapsed; nothing was noticed till a few days later, when he got feverish and apparently jaundiced, and passed urine containing much urobilin. The reading of the case was that there was probably a slipping of a ligature and a large hæmorrhage. Another similar case is the following:—A woman who had not menstruated for several months had sudden pain in the abdomen, and became suddenly collapsed. It was supposed that she had an intra-abdominal hæmorrhage, such as a pelvic hæmatocele or ruptured tubal gestation; the abdomen, however, was not opened. A few days afterwards she became apparently jaundiced; she passed this dark urine containing a large quantity of urobilin, and subsequently got well; and although the diagnosis was not confirmed, it is probable that some hæmorrhage occurred.

There is this same condition of urobilinuria to a slight extent in many diseases, and more particularly in febrile diseases; but diseases which are associated with disintegration of the red blood-corpuscles to a considerable extent, as those just mentioned, are the maladies in which marked urobilinuria is most apt to occur. I might give you a long list of diseases in which urobilinuria occurs, but it would not serve any practical purpose; nevertheless, I would mention especially purpura simplex and purpura hæmorrhagica, and here also the urobilinuria is dependent upon the disintegration of blood-pigment. Urobilinuria is readily recognised; the urine may look as if it contained bile, but the bile tests do not come off properly, and you may notice that on adding nitric acid, instead of the proper play of colours beginning with green (which

is the important colour in the play when bile is present), there is an imperfect play of colours, but there is no green colour, and this should raise your suspicion as to whether bile is really present, and then you must use the spectroscope and seek for the presence of the urobilin band. Bile pigment does not give this band.

Hæmatoporphyrinuria.—There is a small amount of hæmatoporphyrin in the urine normally, but you will find large quantities under certain conditions. The administration of large doses of sulphonal to patients will sometimes make them pass urine of a red colour. Hæmatoporphyrin imparts a very remarkable colour to the urine; it is not quite a cherry colour, it is more of a claret. This hæmatoporphyrinuria is seen especially after the administration of sulphonal, but it also occurs in disease, thus it is sometimes seen in rheumatic fever, in cardiac disease, and in Addison's disease. The causes and nature of it are obscure, but it has been supposed to be dependent on internal hæmorrhage, and especially on hæmorrhage into the alimentary canal. Hæmatoporphyrin can be extracted from the urine by the use of the method described above for the extraction of urobilin, and like urobilin it is identified by its spectroscopic characters.

Uroerythrin is increased in all febrile states, and is a pigment which is familiar to all as colouring urates pink.

The chromogens present in the normal urine are skatol and indol compounds and their allies in the form of aromatic sulphates. These bodies are formed in the intestines, and I will digress here to point out a curious function of the kidney. As every one is taught, the kidneys and the skin have a certain relationship, but the kidneys and the intestines have one also. It is very remarkable that these aromatic sulphates, which are more or less products of intestinal putrefaction, should not be excreted by the fæces, but that they should be absorbed from the intestines and excreted by the kidneys. It draws your attention to the fact that substances are sometimes excreted by what, at any rate, seems a very roundabout channel. Some physicians have thought that uræmia may be really intestinal poisoning, since if the functions of the kidneys are interfered with, the patient may be poisoned from toxic bodies formed in his own intestine. These aromatic bodies tend to be excreted

in the urine in far greater amount than normal when the putrefaction in the intestines is increased. In typhoid fever the intestines are swarming with the *Bacillus coli-communis*, and there is, at any rate, considerable decomposition of proteid matter, and such patients pass dense, high coloured, brownish-red urines, which are loaded with these skatol and indol pigments. Another instance of the same thing is where there is increased intestinal putrefaction owing to intestinal obstruction; and even people who suffer from constipation only have yellow conjunctivæ, and pass dense, high-coloured urine.

In cases of intestinal obstruction there is an abundant excretion of these skatol and indol compounds, and this is especially the case in chronic obstruction, where the colon may dilate to a great size, and be full of liquid fæces undergoing decomposition, and hence it is easy to understand that the absorption of these pigments is considerable. Peritonitis is an instructive example of the same thing, and in this disease you get these substances in the urine, since, owing to the paralysis of the bowel, there is a considerable amount of putrefaction of the intestinal contents. These skatol and indol compounds are also present in the urine in increased amount when there is extensive suppuration in some parts of the body, as in empyema. Indican is a body of this type, inasmuch as indican is indoxyl potassic sulphate, and the term indicanuria is given to the condition where indican in excessive amount is excreted in the urine. Indican is formed in the alimentary canal from indol, and this is united with sulphuric and glycuronic acids as an aromatic compound, and is excreted by the kidney. In conditions where the intestinal putrefaction is increased, indicanuria is apt to ensue. Patients with intestinal obstruction may actually pass a urine which although not blue at the time of passing, becomes blue from the oxidation of indican to indigo, so that indicanuria has the same clinical significance as the presence of excessive amounts of skatol pigments in the urine. It points to the increased breaking down of proteid matter, and, although usually intestinal in origin, yet indicanuria may result from absorption from large purulent collections in various parts of the body. It is, however, more common as a result of intestinal obstruction and putrefaction, and one of the best instances is cancer of

the rectum, since this disease runs a chronic course, and therefore you have every opportunity for increased excretion and absorption. The presence of large quantities of indigo in the urine is easily detected; you take the urine and add to it about a quarter to a third of its volume of hydrochloric acid, and some calcium hypochlorite, let it stand for twenty-four hours, and then the blue scum is observed on the surface. Indigo is sometimes present in such a large amount as to form a calculus, and there is a celebrated specimen in St. Thomas's Hospital Museum.

There is a rare pigment occasionally present in the urine called melanin, and to this condition the name melanuria is given. It is a condition of some interest, but not of any great practical moment. These patients pass a urine which is the colour of *café au lait*; it is often muddy-looking, and when you proceed to test this with nitric acid it goes absolutely black, as you see in this specimen that I obtained some years ago. This is very characteristic of melanuria; it is extremely rare, and I have only seen this one case. The patient had had the eyeball removed for melanotic sarcoma, and came under observation some years afterwards with a number of large tumours in the liver, probably melanotic sarcomata. Melanuria is sometimes seen where you cannot recognise any cause, and it may be the only departure from health; but more usually it is seen in patients with melanotic sarcoma, but you must please understand that it by no means occurs always in melanotic sarcoma. Melanotic sarcoma is not a very rare disease, but melanuria is more rare than melanotic sarcoma. I only mention it for the sake of completeness.

The presence of *blood* in the urine of course alters the colour very considerably. First of all you must make the distinction between blood-corpuscles in the urine and blood-pigment without corpuscles: the former is usually spoken of as blood in the urine, and the latter as hæmoglobinuria, though as a matter of fact the pigment is in both cases hæmoglobin. Blood-corpuscles are found in the urine in a great number of conditions which are very roughly classified as follows:—Blood in the urine as the result of acute congestion of some part of the urinary tract, as the result of passive congestion, as the result of ulceration, and as the result of rupture into the urinary tract. For instance, you may have hæmaturia from the acute

congestion of the kidney that results from acute nephritis, or from acute cystitis, or acute urethritis; these are all instances of blood in the urine from acute congestion. As regards passive congestion it is well known that blood may appear in the urine as the result of passive congestion of the kidneys, as in cardiac diseases, or, as in some cases of engorged prostate, where there is a long-continued distension of the veins of the prostate. Blood also results from ulceration, as in tuberculous pyelitis or in tuberculous and malignant disease of the bladder. Another cause is from rupture; this is rare, but it is conceivable, and recently I saw a case where hæmaturia apparently resulted from an aneurysm of the renal artery projecting into the renal pelvis, but this cause is unlikely. Now as regards the amount of blood, there may be only a few blood-corpuscles or there may be so much blood that the blood coagulates in the bladder in one solid clot; as extreme instances one may mention a tuberculous kidney, from which you may have a very small amount of blood, or a villous tumour of the bladder, where the amount is often very large. A stone in the kidney may cause very scanty hæmorrhage or it may be profuse, and occasionally in tuberculous kidney there is very profuse hæmorrhage. A more important question than the amount of the blood is the source of the blood, and you remember the fundamental point, that if the blood is of renal origin (and it is convenient to include the hæmorrhage from the renal pelvis under the heading of renal hæmorrhage), the blood is intimately and uniformly mixed with the urine. On the other hand, if the blood come from some other part of the urinary tract, it probably is not so intimately mixed with the urine, but it may be. Thus in hæmorrhage from the bladder the blood is not necessarily mixed with the urine, though it may be, and the usual typical bladder hæmorrhage is characterised by the fact that the first portions of the urine are clear, and the latter portions blood-stained; that is the typical condition in cases of hæmorrhage from the bladder, including the prostate, but it is a rule liable to many exceptions. As the bladder contracts down on the diseased structure or surface, the hæmorrhage is produced, and therefore the urine voided during the latter part of the micturition contains more blood, and it is a very characteristic thing of prostate hæmor-

rhage that the very last portions are full of blood. Occasionally in prostatic and urethral hæmorrhages, the first portions of the urine contain most blood; it is a thing that one reads about but does not often see. To consider more in detail some of the causes of blood in the urine: in acute nephritis you have hæmorrhage from the kidney; as a rule the hæmorrhage is not very abundant, because you must bear in mind that in acute nephritis the quantity of urine passed is small, so that the hæmorrhage as a rule is not very profuse. The hæmorrhage accompanying infarction of the kidney is also usually scanty. In pyelitis, the acute congestion of the pelvis of the kidney results from the inflammation of the pelvis, accompanying the presence of stone or tubercle. In passive congestion, as, for instance, in heart disease, the blood is usually small in amount, the urine is often only just tinted.

The hæmorrhage that occurs occasionally in the granular kidney is a difficult form of hæmorrhage to classify. These patients are liable to various hæmorrhages; they readily bleed from the nose, and every now and then they bleed copiously from the urinary tract, and the amount of blood lost is often large. Many of these patients are fairly well, they have not complained much, and they may come to the medical man for the profuse hæmorrhage, and it may be thought that they have malignant disease of the kidney, but they really have a granular kidney. These very profuse hæmorrhages in cases of granular kidney are probably hæmorrhages from the mucous membrane of the renal pelvis, and not from the true kidney substance. Another disease which produces very copious and alarming hæmorrhage, of which the mechanism is not well understood, is purpura hæmorrhagica, where you get very severe bleeding, and it is known that this hæmorrhage is from the pelvis of the kidney, since on post-mortem examination a remarkable condition is found. On slitting up the kidney and the ureters the kidney substance is found healthy, but the whole mucous membrane of the ureters and renal pelvis is raised up from the presence of blood-clot underneath. The hæmorrhage undoubtedly occurs from the pelvis. It is frequently not recognised that the source of the bleeding is the renal pelvis and not the kidney substance, and you must bear in mind that you may have a dangerous and even a fatal hæmorrhage from the pelvis of the kidney in cases

of purpura hæmorrhagica. Hæmorrhage from ulceration is seen especially in calculous and tuberculous pyelitis, and in malignant disease of the kidney.

There are two diagnostic problems connected with hæmaturia, one to diagnose the presence of blood in the urine, and the other to determine its source. There is nothing to equal the microscope in determining the first point, every other test is a long way behind it, there is no test for blood so delicate as seeing the blood-corpuscles. One may not be always familiar with the appearance of the blood-corpuscles as seen in the urine, their colour is not very striking, you may easily think it is a pus-cell; and in the second place it may be profoundly altered, if it is a dense urine it is crenated, and may then be mistaken for a pus-cell; on the other hand, if it is a dilute urine it may be swollen out so as to be almost bursting. As regards the other tests, speaking with all due deference to the spectroscope, it is, on the whole, of very little use, except in the case of a man who has spent a great deal of time with it, which the ordinary medical practitioner has not done. It is a very good thing to use if you are familiar with it, but otherwise it is not an instrument to use for the recognition of blood in the urine in the form of blood-corpuscles. If you use a spectroscope the point is this: you do not rely on the presence of the two oxyhæmoglobin bands, but the point you rely on is the replacing the two bands by a single band on reduction; you must combine the chemical with the physiological condition. There are many substances that yield bands resembling the oxyhæmoglobin bands. The guaiacum test is a very good one, though the chemists sneer at it. A large number of bodies yield the guaiacum test, but they are not bodies which are usually found in the urine, and consequently it is not a bad test; the only point about it is, if you want to do it properly you should put your tincture of guaiacum in the urine, shake up the two and then pour the ozonic ether on the top, so as to get a good line of demarcation between the two. Iodide of potassium will give a reaction something like that yielded by blood, with the guaiacum test, but the colour is more green and diffuse, and the blood one is more blue. The hæmin crystals test is a very good test. These are the principal methods by means of which you detect the presence of blood. There are none of them so good as the microscopical test.

A CLINICAL LECTURE

ON SOME

SKIN ERUPTIONS PRODUCED BY DRUGS.

Delivered in the Department for Skin Diseases, Sheffield Royal Hospital, July 23rd, 1897, by

ARTHUR J. HALL, B.A., M.B. Cantab., M.R.C.P.,

Physician to the Hospital; Lecturer on Physiology.
University College, Sheffield.

GENTLEMEN,—I propose to consider to-day certain skin eruptions which do not form part of any general diseases, neither are they due to any constitutional affection of the patient; they are simply visible effects produced in the skin by certain drugs which the patient has used for remedial purposes. Examples of these so-called drug rashes will from time to time come before your notice; but, as you can readily understand, it is impossible to collect together many varieties at one clinical lecture. It is not an infrequent occurrence to find a drug rash mistaken for something else, and as the mere cessation of the offending drug will speedily cause the rash to disappear, whilst, on the other hand, a continuance of the drug will frequently aggravate it, it is very important that the true cause should be speedily recognised. It is also of importance because you will find many patients who particularly resent having a skin eruption thrust upon them so to speak, not only because it is a visible disfigurement which their neighbours can see as well as themselves, but also because there is amongst most people a kind of stigma attaching to skin diseases which does not apply to diseases of many organs which are far more serious. To avoid any such oversight it is a good routine rule to always ask patients suffering from skin diseases if they are or have recently been using any drugs at all. Apart, however, from the patient's own feelings, the occurrence of a drug rash sometimes, not always, is a visible evidence that the drug is being given to excess, and other symptoms of poisoning accompany it; sometimes the accompanying symptoms are serious, and may actually endanger life if the drug is continued, whilst in other cases the presence of the characteristic rash does not necessarily contra-indicate a continuance or even an increase of the drug.

Another important point to bear in mind is that certain patients will get a rash from almost any drug that is capable of producing rashes, even when given in comparatively small doses. I have seen a case in which small doses of potassium bromide produced well-marked bromide rash, small doses of iodide of potassium a severe iodide rash, whilst ordinary doses of belladonna produced symptoms of belladonna poisoning almost directly; and when patients tell you that they are very susceptible to certain drugs, it is always well to heed them and proceed cautiously.

The drug eruptions I shall speak of to-day have all been under my care in this department, and some examples of them I shall be able to show you as I speak about them.

I shall divide drug eruptions into two classes:

Class 1.—Eruptions produced by the presence of the drug or some compound of it in the circulating media.

Class 2.—Eruptions produced by the presence of the drug on the external surface of the skin itself.

In considering Class 1, it is evident that the drug may have been absorbed into the circulation by various routes, either—

1. By the stomach.
2. By hypodermic injection.
3. By some other mucous surface, such as the vagina, bladder, or intestine, or
4. By the broken or unbroken skin.

Of these classes the first is the more common, and of it the first division, but the other divisions of Class 1 are more likely to be overlooked, and are of considerable importance, as the absorptive surface is often large and the drug is absorbed unchanged by the action of any digestive juices.

Before commencing an account of the rashes *seriatim*, I would remind you that in the case of two or three drugs there is usually a characteristic eruption which may be diagnosed with comparative certainty at sight; but the rashes produced by most drugs are so various and differ so greatly in different individuals that the most experienced skin specialist could not in most cases from merely looking at the rash itself, at once say, "This is a quinine rash, this is an arsenic rash," &c., &c. Each drug is capable of producing a large variety of rashes depending for their position, extent, and character on the individuals in whom they are

produced. A very curious example of what I have just said occurred in this room one week ago. As you know, I was intending to give this lecture last week, but owing to you expressing a wish that I would postpone it in order that you might witness an unusually interesting operation which was fixed for the same hour, I did so. Unfortunately for you, directly after you had gone, a woman, whom I had seen as an out-patient two days before, came into the room, saying she had "begun with a rash." It was most striking in appearance, being very extensive and consisting of bright scarlet erythematous patches of large size and various shapes, which in many places had become vesicular, especially so on the lobes of the ears, which were covered with tense blisters, and were the only parts of the head and neck affected. Now, as you will hear later on, iodide of potassium frequently produces a very characteristic eruption, the well-known iodide rash, which is neither erythematous nor vesicular, and yet this woman was undoubtedly suffering from a more rare form of rash produced by taking iodide of potassium, which I had prescribed for her two days before. The rash which had appeared after the second dose began to disappear within a few hours of leaving off the medicine. I am confident that no medical man, if shown that case without being told anything, could have said or would have been warranted in saying that it was produced by iodide of potassium; he might have eliminated other causes and suspected a drug rash, but that would have been the limit of his diagnosis. What I want you therefore to take to heart is this, by closely observing drug rashes learn the general phenomena which characterise almost every drug-rash, viz. (1) Unusual positions affected; (2) Polymorphism or variety of lesions at the same time; (3) Frequent angio-neurotic character; (4) Presence of other symptoms of poisoning; (5) Abatement on withdrawing the drug; and (6) Possible reappearance directly it is renewed. You will frequently be seeing surprising drug rashes, and they are unfortunately so often like those due to specific fevers that however careful you may be you will be deceived; but there is usually something in the symptoms which will make you suspect the drug if you go into it thoroughly.

The two commonly used drugs which produce characteristic eruptions are the bromide and iodide

of potassium, and examples of these are so common that you will see many cases of "bromide" and "iodide rash." Potassium bromide, which is so much used in the treatment of epilepsy, frequently produces an acne-like rash, chiefly on the forehead and face and shoulders, but sometimes extending further than this, and differing from acne in the absence of comedones. It is a papular, tubercular, and pustular eruption, being primarily an affection of the sebaceous follicles. Iodide of potassium produces a very similar rash to the above, but in the cases I have seen it has been as a rule more extensive, and the tubercular swellings larger and more inflamed. Copaiba frequently produces a roseolous rash, which is known as *roseola balsamica*; its distribution may be over the whole surface, but it particularly picks out the neighbourhood of joints; the shades of colour on different parts of the body vary considerably. It consists of bright red spots running together into patches. There are other forms of eruption, but this is the commonest one.

Arsenic, which is so largely used for the internal treatment of skin diseases, may itself produce eruptions, as the following case will show. W. M—, a baker, æt. 68, was admitted into this hospital suffering from chronic eczema; he was treated with local applications and gradually increasing doses of liq. arsenicalis. On September 19th he had ℥xv per day, on the 23rd ℥xxiv, on the 30th ℥xxvij. The original complaint had almost disappeared on October 1st, when he developed large erythematous pustules on the legs and back, and later on the arms and hands. They were very painful and inflamed. On October 3rd the liq. arsenicalis was increased to ℥xxx per day, and on October 7th to ℥xxxij. The pustules got worse, and on October 8th he complained of shivering and was slightly delirious. The arsenic was at once stopped, and the pustules rapidly disappeared.

Herpes is a not uncommon form of arsenical rash.

Quinine occasionally produces skin eruptions, most commonly erythematous, but more rarely there may be wheals, bullæ, petechiæ, or even gangrene. The following notes of a case of quinine rash show how the erythematous form may be difficult to distinguish from a specific fever.

C. T—, boy, æt. 4 years, was admitted into the

boy's ward on July 1st, 1895, suffering from follicular tonsillitis. There were large yellow patches on the right tonsil and soft palate; enlarged glands. No dyspnoea, no albuminuria. A bacteriological examination did not show any diphtheria bacilli. The throat cleared up very quickly. The boy was put on quininæ sulphatis gr. j three times a day. He was apparently quite well on July 12th, when a rash was noticed on his face, hands, and feet. There was no sore throat, no coryza, or catarrh. Tongue clean. Temperature 100°.

Over face, legs, and arms there was a papulo-erythematous rash, forming patches of various shapes and sizes, not at all crescentic; felt rough. Some slight spots on the body. The boy seemed quite well himself, and as I was very uncertain as to whether this was a quinine rash, or whether he was beginning a specific fever, I ordered a double dose of quinine to be given in the afternoon. At 10 p.m. the temperature had reached 104°; the rash was more fully developed, but still chiefly on the face, arms, and legs; the tongue dry, conjunctivæ inflamed; on parts of the cheeks the patches had run together so as to form œdematous-looking swellings. The quinine was now left off, and some calomel given. Next day, July 13th, the temperature was normal, the boy seemed well, the patches on face and upper arms and legs had become confluent. The next day it had begun to fade, and on July 16th had almost entirely disappeared from the face, whilst the rest was fast disappearing.

I have no doubt that was a quinine rash, although the high temperature is not a usual symptom. It is sometimes almost indistinguishable from scarlatina, as the fauces are frequently affected.

Salicylate of soda, although frequently producing symptoms of poisoning—dizziness, noises in the ears, delirium, &c.,—does not often, in my experience, produce an eruption, but I have here to-day a patient in whom I believe it has done so. This patient, C. S—, a man æt. 51 (O.P. 1032, April, 1897), was admitted as an out-patient in April, 1897, suffering from rheumatism of the right shoulder-joint. He was at first put upon iodide of potassium without benefit; afterwards, however, he was given sodium salicylate 10 grains three times a day. On June 2nd, 1897, he came up complaining of a rash which had appeared on the face and legs. On July 2nd it was limited to the forehead and sides

of the nose and cheeks, and the inside of the thighs. It was a bright scarlet-erythematous rash, with raised tuberculated lumps here and there of a purple colour; there was one small pustule close under the edge of the hair on the forehead, everywhere else the rash had the characters described above. There was a fine brawny desquamation on the nose and forehead. On the thighs the tubercles were more marked than on the brow. On July 9th I ordered him to leave off the medicine, and the rash quickly faded.

Various rashes have from time to time been found to follow the use of this drug, and although they are very varied in character they should be borne in mind.

Boracic acid and borax when taken in the stomach may produce eruptions. They may, and undoubtedly do, produce eruptions when absorbed into the circulation through a wound, or when used for washing out cavities, such as the pleura, peritoneum, &c. Boracic acid in the form of ung. boracis or ung. ac. borici (the two are identical) is very largely used at the present time as a dressing for burns, ulcers, &c. Boracic lint, that is lint impregnated with boracic acid, is also largely used for dressings, fomentations, &c., and I have seen recently in this hospital, and heard elsewhere of boracic acid eruptions in no few numbers. The reason boracic acid has been and is so largely used is that it approaches the ideal form of antiseptic dressing, for it prevents or tends to prevent septic action, and is not irritating. But it should be remembered that when a very large surface, such as a burn, is covered with boracic acid in solution from morning till night and night till morning day after day, a very large quantity may be absorbed by the blood-vessels and lymphatics, and may produce very serious symptoms of poisoning.

The first time I ever saw this eruption was in a case which was admitted into the wards under Dr. Sinclair White for burns. The boy (P. W.—) was aged 8 years, and had been extensively burnt on the trunk and limbs on September 1st, 1895. He had gone on well with some slight pyrexia until September 5th, when I was asked to see him on account of a rash which had that day appeared all over the body and limbs. It was of a bright scarlet colour, erythematous, punctiform in places, in others running into irregular patches, giving a mottled appearance to the skin. The palms and

soles were intensely red, looking as though they had been painted over with red paint. At the edges of the burns the erythema was well marked. There was no sore throat, no catarrh, the tongue was dry and glazed; temperature 102°. No medicine was being taken internally, and the wounds seemed perfectly healthy. I was asked to say whether the case was one of scarlatina or not. I did not think that it was for the following reasons: (1) There was no sore throat; (2) The temperature had not suddenly risen; (3) There had been no vomiting or rigor; (4) The intensity of the rash at its onset. I thought it was possibly a quinine rash, and made careful inquiries as to drugs, but, as I told you before, with negative results. The boy got steadily worse, the rash became more vivid, the temperature fell, the conjunctivæ became inflamed and covered with secretion, and the boy died semi-delirious on September 9th. An autopsy was made the next day, and nothing abnormal found except a few petechiæ at the pylorus and a commencing duodenal ulcer. It was not until after this that I bethought myself of the possibility of the boy having really suffered from boracic acid poisoning, and I looked up the literature of the subject. I there found that quite a number of cases of boracic acid rash with more or less serious toxic symptoms had been recorded, and I have no doubt that this was such a case.

Since then I have seen several cases of scarlatini-form rash following the use of boracic acid as ointment or fomentation; in one case of burn the patient died six days after the appearance of the rash with symptoms almost identical with those of the case I have just related to you, and this although the ung. boracis was immediately withdrawn on the first appearance of a rash. In this case I was able to demonstrate the presence of boracic acid in the urine, showing that it had been absorbed into the system. From these and other cases I am convinced that boracic acid is absorbed from a broken surface, and that it does frequently produce an erythematous rash. Sometimes that is all, the rash fades although the drug is continued; but sometimes other and serious, even fatal symptoms, may and do occur, especially if for some reason or another there is an inability to get rid of the excess of boracic acid from the system. Do not suppose that I wish you to infer that all scarlet erythematous rashes following burns are due to

boracic acid poisoning, for scarlatina undoubtedly occurs not infrequently after burns and surgical wounds. We had a case of the former in our Nursery Ward only a few weeks ago,—in which the nurse got it too. Moreover, it sometimes runs an anomalous course; but whenever you see a doubtful case, be sure to eliminate boracic acid as a possible cause.

The last drug I shall call your attention to comes under the head of Class 2—oil of Cade. It is a very useful tar preparation, but sometimes when applied to the skin produces an inflammation of the hair follicles, a *folliculitis*; this is a form of so-called tar acne, which workers in tar are liable to. In this case the oil of Cade ointment quickly cured the rash on the legs for which it was applied, but produced an uncomfortable folliculitis for which the patient went on rubbing in the ointment daily and made gradually worse. In this case, of course, the irritant was entirely extra-vascular.

As regards treatment, of course the simplest proceeding is to stop the use of the drug; but sometimes it is very desirable to continue the drug, for example, bromide of potassium in epilepsy, iodide of potassium in syphilis, quinine in ague. In the two former liq. arsenicalis in small doses added to the medicine is sometimes, but not always sufficient.

Sometimes, curiously enough, an increase of the dose of the drug will suffice, but in the case of quinine rash it is usually necessary to stop it altogether, and in boracic acid rash also it is certainly safer to do so.

DEMONSTRATION OF CASES

At the Monthly Meeting of the North-West London Clinical Society, held at the North-West London Hospital, June 16th, 1897.

Dr. CUBITT LUCEY in the Chair.

Cases of Appendicitis.

Mr. JACKSON CLARKE spoke on two cases of appendicitis of the relapsing type. In the first he did the usual operation. In opening the peritoneum, which was considerably thickened with the whole

of the abdomen considerably infiltrated, he came upon the omentum, which was adherent to everything. After getting through the adhesions carefully and lifting the omentum off the cæcum, and dividing other adhesions, he saw the cæcum and the end of the ileum, but no appendix or trace of it was in view. Before operation he had felt a mass deeper than the omentum, and he thought probably the appendix lay in that mass. He identified the appendix lying along the false pelvis towards the brim, and with his finger-nail he dissected out its remains from the middle of a mass of fibrosis; there was a faecal concretion close to the remains of the appendix. When cutting a section of it afterwards and examining it microscopically, he noticed that the epithelium of the tube had been destroyed by inflammation. The faecal concretion would have no doubt lighted up a fresh attack. The point in the case was not whether it was desirable or not to operate, because of that there could be no doubt, but as to the technique of the proceeding. The patient was a man aged 25, who did very well for six months, but at the end of that time he had been enjoying himself a little too much, both in muscular exercises and in revelries, and an abscess formed immediately beneath the scar, which opened and discharged without any further trouble. Since that happened, some years ago, the patient had not had a fresh attack. He, Mr. Clarke, attributed the abscess to irritation caused by the sterilised silk sutures he used at the operation. From the way he had to dissect out the embedded appendix he felt that there might have been some contamination of one of the sutures, and the extra exercise might have determined the formation of the abscess.

The appendix from the second case was passed round for inspection; it was considerably shortened and thickened, and with it was some thickened and greatly infiltrated omentum. The patient was a lady aged 29, and her case was also of the severe relapsing type. As she was a singer it was desired to leave her without any belt or artificial support to suggest that she had any abdominal weakness likely to interfere with her vocation. In this case he made use of the incisions recommended by Mr. Battle, and found that they worked admirably, both at the time of the operation and subsequently. The patient called upon

him eleven weeks after the operation, and stated that she was perfectly well. In this case also there was a little complication. The tip of the appendix had curled round towards the front of the cæcum, and between it and the omentum above and the cæcum behind was what had been an abscess cavity, in which he found only granulation tissue and some necrotic shreds, which he stroked away with gauze tampons, wrung out of 1-20 carbolic lotion. After removing the appendix and examining the cæcum he found there was a spot of weakness in the wall of the cæcum, which was practically only closed by a mucous membrane. It was so weak that he put in a few Lembert sutures, indenting the weak area. This was what one would call a "cold" case, all active suppuration and all traces of it having ceased. Though the operation adopted in this case was longer, it was not very much more difficult than the ordinary one, but it required a little more anatomical familiarity. He (Mr. Clarke) considered that this particular method of Mr. Battle's should be limited to the "cold" cases where all active suppuration had ceased. In cases where there had been much œdema of the abdomen, and where one would expect to meet with very tough adhesions in the neighbourhood of the cæcum, he thought the ordinary direct incision running obliquely out over the outer part of Poupart's ligament would be safer from the operative point of view. Again, in weakly patients he thought they should sacrifice the final result for the greater safety of the patient during the operation. For cases where they wanted a very strong abdominal wall without any trace of hernia, and where they did not expect to meet with any extraordinary amount of adhesions in the abdomen, he could certainly say that he thought the operation a very valuable one, and he was very well satisfied with the result in this particular case.

Mr. BATTLE said he was very glad to hear Mr. Clarke had found his method of operating so far a success in actual practice. He (Mr. Battle) had employed it on many occasions; in fact, he had always used it for the removal of the appendix in what might be called relapsing and recurrent cases. He advised it in the first place because he had not seen very good results in several patients who had been operated upon by other methods. The first private case on which he was called to operate was

one where the patient's livelihood depended upon his ability to take active exercise; in fact, he was applying for a post which involved a great deal of riding on horseback, and any abdominal weakness would have prevented his filling the post. He had always found that the operation had answered his expectations of it. The principal point about the operation was that the deeper section of the abdominal wall was carried more towards the middle line than in the ordinary operations,—that is, the skin incision commenced fairly well in the line of the linea semilunaris, and then the further section of the tissues in front of the appendix was carried one or one a half inches towards the umbilicus, further from the appendix, so as to open the front of the sheath of the rectus. The rectus itself was then drawn towards the middle line, and the posterior layer of the sheath was cut through with the peritoneum. Afterwards the removal of the appendix was effected in the ordinary way. Then the tissues behind the rectus were sutured, and the rectus was allowed to come back into its proper place. Afterwards the muscle and the sheath in front were combined in one line, and the rectus was thus interposed as an effective barrier against hernia of any part of the wall which might otherwise have been weak. He first did the operation for an ordinary relapsing or recurring appendicitis, in which the appendix could be felt from the front, and seemed easy to get at. This was found to be the case. The question which had to be considered then was whether by means of this operation the seat of the disease could be got at as readily as by any other operation. He had found it answer this purpose extremely well. The linea semilunaris was not so near the umbilicus as compared with its position towards the anterior superior spine as people thought. By means of this operation he had been able to get at the appendix in every case, and in all except one he had been able to remove the appendix through the primary incision very easily. In the case which formed the exception, although the patient had had several attacks of appendicitis, the appendix was tucked up behind the cæcum, and could not be differentiated. He could only have got to the appendix by dividing the peritoneum outside the cæcum and dissecting the cæcum up. He did not attempt to remove it because he did not think the patient was in any danger to life from this unusual

position. Of course the operation did take longer because the layers had to be separately sutured, but he found that by putting in continuous instead of interrupted sutures the time was very materially diminished. He was interested in Mr. Clarke's statement as to his finding a loose concretion in one of his cases, because it was most important to remove any concretions. A concretion anywhere in the region of the appendix, whether inside or outside, was a source of danger, and it was necessary to get it away. He (Mr. Battle) had had one interesting case in which there was an abscess about the appendix. When the abscess was opened and fully evacuated, nothing came away. There was suppuration around, and everything was matted. The patient rapidly recovered up to a certain point, but a sinus remained which would not heal. The question arose as to the nature of the sinus—why it did not heal. A probe passed into the sinus went as far as the wall of the rectum. Later, as there was no improvement, he thought there might be a concretion in the sinus. He therefore sounded along the whole of the sinus with a bullet probe, and at one part struck what he believed to be a concretion; then with the alligator forceps he managed to get away the chief part of a small fecal concretion. As there was considerable bleeding and pain he left it for a day or two, and then the house surgeon introduced the forceps and removed the rest of the concretion. The sinus then healed up.

Case of Locomotor Ataxy with Unusual Symptoms.

Dr. HARRY CAMPBELL showed a man the subject of locomotor ataxy. The interest of the case consisted in the fact that the symptoms had shown themselves in the upper extremities before the lower. The symptoms referable to the upper extremities were decided. There were anæsthesia, a certain amount of inco-ordination, and lightning pains; whereas the symptoms referable to the legs were slight. There was, however, loss of knee-jerk, and there had been indications of lightning pains in the legs, and perhaps some slight commencing anæsthesia also. The patient could not balance himself quite so well as a normal individual could, but this was scarcely perceptible. When the patient came under observation the first thing he complained of was numbness of the

fingers; a pain shooting down his arm from the elbow to a spot in the palm of the hand and playing around that spot, the patient likening the feeling to that which would be produced by injecting hot water along the affected part. If there was a scratch or sore in the hand the pain would shoot down to that part and play around it. He had unmistakable areas of anæsthesia, and there was a delay of pain sensation as compared with touch sensation; it would be noticed that when pricked with a pin he almost immediately indicated that he felt the touch, and about two seconds afterwards would show that he felt the pain. He presented another curious phenomenon, namely, great sensitiveness to heat. If he put his hands into moderately warm water, as soon as the hands touched the water he would notice the warmth, but when they had been in two or three seconds he would manifest symptoms of obvious pain. He also had a tendency to girdle pain, and had typical Argyll-Robertson pupil—failure to react to light, but ability to react to accommodation. Eight or ten years ago the patient seemed to have indulged very freely sexually, and was capable of unusual sexual feats. That was known to occur in the early stage of locomotor ataxy. As this was eight years ago, it impressed upon them the insidious progress of many of these chronic nerve diseases; the early symptoms probably started long before they were recognised by either patient or physician.

Dr. GUTHRIE remarked that the lower extremities were certainly usually affected first in tabes, but there seemed no reason why a lesion which affected the higher part of the cord, and therefore the upper extremities, should not occur first. Usually he believed the lesion was in the upper and mid-dorsal region, but in the present case it had doubtless occurred higher up. The delay in sensation was to him very unusual, for he had never met with it, though it had been described in Hilton Fagge's 'Medicine,' on the authority of Erb, Cruveilhier, and others. In one case sensation to pain was not perceived until nearly half a minute after the prick which produced it; there was also delayed sensation of heat and cold. When Hilton Fagge wrote that, he (Dr. Guthrie) believed syringomyelia was hardly known, and therefore it could be suspected that some of his cases regarded as locomotor ataxy were really syringomyelia. The present case was no doubt one of pure locomotor ataxy.

Dr. BOUETING said he would like to know whether Dr. Campbell believed this case to support the recent doctrine of physiologists that there are special nerves of pain. It was rather remarkable that while the patient felt heat immediately, the pain which followed on the heat took the same time as the pin-prick. He would also like to hear if there were any history of prior syphilis in the case.

Dr. CAMPBELL, in reply, said there was a history of syphilis in the case. It was fairly well established by physiologists that there were special nervous mechanisms for touch sensations, for heat sensations, and for cold sensations, as well as for pain sensations. It had been demonstrated that there were special organs in the skin devoted to touch, heat, cold, and pain sensations. There was once a tendency to regard pain as an exaggerated touch sensation, but that was not so; pain was a special sensation.

Early Condition of Leontiasis.

Mr. BATTLE showed a boy *æt.* 15, the subject of leontiasis. The patient presented one of the most interesting examples he had seen. He came to the out-patient department of the Royal Free Hospital a week ago complaining of a lump on the back of his head. The right side of the scalp hung down and looked as if there were some *nævus* growth there. It was said to have been present eight years, and to have come on after scarlet fever. It was a soft, hardly-defined tumour, which felt as if it could be emptied, but on close pressure it was evident that no change was produced. Nothing in the nature of irregular veins could be felt. On asking him if he had anything else, he said he had another lump on the left side of the head, which had followed injury by a stone. The hair on these tumours—for there were two—was partly gone. On asking him if he had anything else, he said he had a tumour beneath the left breast. This resembled a *nævus* very closely, because there was irregularity in the feel of it, and it had a bluish appearance. The patient then said he had another lump on his back; and on looking a number of tiny tumours were seen dotted about, some of which were pigmented. The size of these varied from that of a millet seed to a horse-bean; they were soft, and distinctly different in texture and feel from the surrounding parts. The same con-

dition was present on his chest. He was said to have had lumps over his chest all his life. Points of lesser importance were that he had lines about the angle of the mouth, and the teeth were undoubtedly those met with in congenital syphilitic disease. Later on he believed these various tumours would become pedunculated, and form the condition spoken of in text-books as leontiasis. The tumours were examples of *molluscum fibrosum*. The patient was not of much intelligence, and there was a peculiar odour about the body. The tumours had no relation to the distribution of the nerves. He had removed a tumour of the same kind, but very much larger, at the Royal Free Hospital, and on removal had found a curious finger-and-toe-like condition underneath, like the diseased turnip where it runs to root, and is covered with little lumps almost like potatoes. Microscopic examination by Mr. Shattock showed these growths consisted of simple fibrous tissue, not nerve elements. There were only two or three other cases recorded in which a similar condition was found, and in some of them the tumours were undoubtedly pure neuromata.

Result of Excision of Hip.

Mr. BATTLE also showed a boy, *æt.* about 15, upon whom he had performed excision of the hip-joint. He said he wished members to see the case because it was about the best result of the kind he had seen. The patient had no high-heeled or high-soled boot, there was no rocking from side to side when he walked, and he could easily flex his leg as far as ordinary boys. He was under Mr. Battle's care at St. Thomas's Hospital in September last year. There was a history of four days' acute pain, and the usual signs of acute osteo-periostitis of the right radius. A few days after admission, when he appeared to be improving, his temperature rose again, and signs of suppuration of the left hip-joint showed themselves. It was cut down upon, the periosteum found tripped from the upper end of the femur, and suppuration was present about the joint and the upper part of the neck of the bone. The head and neck of the femur were removed. The patient gradually recovered, and there was only one interruption to progress, namely, after he had left the hospital he came back again in a few weeks with some inflammation about the upper end of the shaft of his femur. This, however, subsided without the formation of a sequestrum.

There was no more shortening than at the time of the operation.

Mr. JACKSON CLARKE thought the first case was not a typical example of fibroma molluscum, which they were accustomed to see either in the shape of pedunculated tumours scattered about the skin or in more diffuse pachydermatous areas. In the latter form one could see great frills of skin, sometimes hanging down around the neck or shoulders. Hypersecretion of sebum could also be seen in these cases, the sebum having a rancid odour; that might account for the odour in the present patient. Sometimes congenital tumour conditions, such as fibroma molluscum, were generalised in the shape of a dermatolysis. He agreed that these tumours usually consisted of lax fibrous tissue, and he was interested in Mr. Battle's remarks regarding the neuromatous origin of the ordinary form of fibroma molluscum. There were other possibilities which might be mentioned in connection with the case before them. Mr. Battle mentioned nævus, and multiple lymphangiomatous tumours. One case of lymphangioma he (Mr. Clarke) had seen was combined with general hypertrophy of one lower limb, and in that case groups of little vesicles appeared twice a year, with considerable pain and swelling in the limb. The case presented a very interesting study in the evolution of tumours.

A good many attempts had been made in cases of congenital dislocation of the hip to procure a good joint by making a new acetabulum and putting a new head of femur into it, but the result had been nothing like so good as in the present case. Though Mr. Battle had removed the head and part of the neck of the femur, there did not seem to be the slightest displacement, and the stump of the neck must be inside the acetabulum. Unfortunately, one could not prognose such perfect results as likely to be obtained after any given excision of the hip.

Cicatricial Closure of Mouth.

Mr. J. G. TURNER showed a woman of middle age who suffered from cicatricial closure of her mouth. About eighteen months ago she had a severe attack of ulcerative stomatitis, which she attributed to drains. This left her with loss of both upper and lower alveolar borders of the left side, closure of the mouth following, so that she

could not separate the teeth at all. There had been ulceration of the cheek, and obliteration of the alveolo-labial sulci on that side from adhesion of the ulcerated surfaces. Before he saw her she had been treated on two occasions by division of the cicatrix, followed each time by considerable hæmorrhage, and in due course by return of the condition. She was also treated with wedges and screws, which were painful to apply, and were found to be quite ineffectual. He found that the skin of the cheek was quite moveable, so that the cicatrix did not involve the whole of the cheek. He etherized her, and attempted to reform the upper and lower sulci. He was fairly successful in the case of the upper, but not in the lower. He separated the inner surface cicatrix and divided the portion he had left horizontally about its middle, so forming two flaps attached by one edge to the remains of either alveolus. These flaps were packed up into the positions of the sulci by iodoform gauze, which served also to control hæmorrhage. She was repacked every day for a week and a bandage put on; and then the house surgeon made some wedges of dry wood, which were cut across the grain, grooved above and below, and these were fitted between the teeth, so that any contraction that had taken place during the week was overcome by swelling of the wood. These wedges were perforated, and a piece of string passed through and tied round her neck, so that she could wear them day and night. At the end of some weeks he (Mr. Turner) made her a permanent wedge, which she now wore during the night only. Moulds were taken, and the wedge was made of vulcanite, the grooves above and below being filled with gutta percha. Members would see that she could open her mouth as wide as any ordinary person need want to, and had been able to do so now for six months. She had had some artificial teeth put in, "with a raised bite," and now wore the wedge at night only. If she did not wear the wedge every night he feared she would relapse. If he had another such case he did not think he would excise the whole cicatrix, but would thoroughly divide it; he would certainly attempt to reform the sulci, though he attached most importance to the treatment by "continuous dilatation" by properly constructed wedges.

NOTES.

The Ear Complications of Influenza.—At the recent meeting of the American Otological Society, Dr. Wells P. Eagleton read a paper on this subject. Of the cases of catarrhal otitis which so frequently complicated influenza, giving rise only to slight pain and transient deafness, little need be said, as they differed in no way from the simple cases, but the cases that went on to suppuration might present one of three conditions that were distinctive, all probably due to the direct influence of the presence of Pfeiffer's bacillus:

1. Distinctive types of hæmorrhagic otitis.
2. Primary mastoiditis or periostitis before the involvement of the middle ear, due apparently to direct infection by the bacillus, and not to extension from the naso-pharynx.
3. Rapid caries and necrosis of the ossicles or mastoid (of very frequent occurrence).

In addition there were minor points of difference from the simple cases, such as the greater severity of the pain and its longer duration, the more frequent persistence of the tinnitus, and the occasional serious involvement of the labyrinth after apparently slight affections of the middle ear.

There were three distinct forms of influenza otitis with hæmorrhages into the membrana tympani, which, however, if properly treated, had not in the author's experience unfavorably affected the course of the disease, although the invasion was apt to be severe.

The presence of the influenza bacillus exerts, says Dr. Eagleton, a very unfavourable influence on the bony structures of the ear, often converting apparently simple cases of acute suppurative otitis into very malignant ones, with rapid destruction of bone, and this without marked symptoms; while in not a few instances the inflammation has developed in the bone itself either as a primary periostitis or a mastoiditis. This tendency to rapid bone destruction should be constantly kept in mind, and can be prevented only by early, and if necessary repeated paracentesis; and even with this some cases will require an early opening of the mastoid to stop the destructive advance of the disease. Failure to perform paracentesis early has in several cases in his experience resulted in caries or necrosis which might have been avoided, and

in one case, that of an old gentleman, ended in complete destruction of both malleus and incus, with almost total deafness of the affected ear.

In the cases, however, in which the mastoid is affected, says Dr. Eagleton, paracentesis alone can accomplish little. Knowing as we now do that in all cases of acute otitis the inflammation never remains entirely confined to the middle ear, but always involves to a greater or less degree the mastoid; and realising the great tendency to rapid caries and necrosis in influenza otitis, we should not hesitate, he says, to open the mastoid early whenever the acute symptoms are not quickly relieved by paracentesis, or whenever protracted and profuse suppuration follows.

New York Medical Journal, August 7, 1897.

The Treatment of Status Epilepticus. By L. PIERCE CLARK, M.D., First Assistant Physician at Craig Colony.

Probably no symptom found in epilepsy is at once so awful in its appearance to the clinician and difficult for treatment as that of status epilepticus. The elevation of temperature, extreme physical prostration, and the corresponding depression upon the whole vascular system renders it a state demanding the most urgent and immediate treatment.

"The fallacy of analogy" applies here with great force whenever we attempt to compare status with a single seizure or any reduplication of a simple seizure. Of course, both conditions are but manifestations of the discharge of nervous energy, but beyond this we cannot hope to draw parallels between the two symptoms.

Since the opening of Craig Colony for admission of epileptics many cases of status epilepticus have occurred. Many remedies have been tried; chloral in large doses (40 to 80 grains) has been given, but little real benefit has been obtained from this drug when it has been given alone and in very large doses. It is a powerful sedative upon the whole vascular system, and especially upon the heart. It cannot help but be productive of great harm when given in very large doses, for at such times the heart is probably labouring under a heavier burden than it is ever called upon to bear in any other affection, therefore chloral should be given with extreme caution.

It is difficult to see how emetics can be of any value, offering as they do great hindrance to normal respiration. Occasionally drugs having this action have been administered, but they have been much more productive of harm than good.

Although chloroform is spoken of by many writers as being only "palliative," yet it can often be used with great advantage if used with caution, and only at such times when the seizures are about to begin. It must never be given at the sleep or stupor stage, as at such times it may cause deep coma and even death. The so-called "palliative treatment" is frequently of the utmost importance; in fact, the palliative treatment is about the only one which is of any great service in treating status epilepticus, therefore chloroform should be tried in all obstinate cases. Morphine has proved of some value when used in conjunction with other drugs, but used singly it shows a very uncertain action in epilepsy, so much so that its benefit or failure in any particular case cannot be proven until it is tried.

Nitrite of amyl is a remedy often spoken of with great favour, but the writer has never seen a case in which its administration was a benefit; on the contrary, he has often seen it do positive harm, even provoking or prolonging an attack. From his experience its disuse cannot be too thoroughly recommended.

It is but fair to say here that in such an institution as the Craig Colony, where the proper food, exercise, and hygienic surroundings are obtained, the manifestations of status in epilepsy are less severe and much more amenable to treatment than cases occurring in a physician's private practice.

The writer has not tried the various remedies recommended for status epilepticus without making some observations and experiments of his own. The prescription which will be given with this article has undergone various modifications from time to time, until it now seems to have become sufficiently crystallised to report for trial away from the Colony. No cases of status have occurred as yet at the Colony where it has not been possible to give this remedy by the mouth; indeed, all medication for that of status should be given at such times when the drug can be taken by the mouth. To anyone unacquainted with the conditions of status the writer cannot urge too strongly

the great necessity of the very earliest treatment possible; half the terrors of status are removed when we recognise that it is a condition demanding immediate action. If the condition is cared for at an early moment the whole organism is in a fair condition to respond to the drugs which may be given, but if delayed, enormous doses of chloral, bromide and morphine may be given without any result whatever. After treatment has been instituted by the prescription which is here given almost all cases of epilepsy have had no seizures following its administration. In a few, two or three attacks have occurred for the first fifteen or twenty minutes after its administration, but no longer. The prescription is as follows:

℞ Tr. opii deod. ℥v.
Potass. bromid. gr. xxv.
Chlor. hydr. gr. xx.
Liq. morph. sulph. (U.S.) 3j.

M. Sig: one dose; repeat in two hours if necessary.

An explanation of the efficacy of this prescription might be hazarded on the following principle: The chloral and morphine are the first to act in their respective order, the chloral as a sedative upon the vascular system, and especially upon the blood supply in the brain; the morphine as a sedative on the nerve cell. Following their immediate combined action we get the slower and more permanent effects of the bromide and opium upon the cerebral centres.

The writer takes pleasure in placing this prescription for status on record, in order that other physicians may try the same to determine its efficacy in all kinds of cases and under different conditions.—*Pediatrics*, August, 1897.

The right way to give Iodine for its therapeutic effects is to dissolve the iodide of potash or of soda in an equal weight of distilled water. Approximately, a minim or drop of the saturated solution is equivalent to a grain of the salt. Direct the number of drops of the desired dose, given in a spoonful of water. Before swallowing the medicine the patient should swallow a small glass of water, and at once after the medicine a goblet of water; the more water taken the better, but never a less quantity than above indicated. Another direction should be that the medicine be taken every four hours, and never close to any meal. The purpose of this is to avoid a contact of the iodine with stomach contents.—*Cincinnati Lancet-Clinic*, August 14th, 1897.

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THE INDICATIONS FOR AND METHODS OF OPERATING ON THE MASTOID.*

BY

H. LAMBERT LACK, M.D., F.R.C.S.,

Assistant Physician, Throat Hospital, Golden Square;
Surgeon to the Ear and Throat Department,
Children's Hospital, Paddington Green.

SUPPURATIVE middle ear disease is one of the most common diseases, and at any time during its course serious symptoms may arise and require prompt operative interference. It is very important, and at the same time most difficult, to accurately define the circumstances in which operation is necessary. On the one hand delay may allow the disease to extend to the lateral sinus, meninges, brain, &c.; on the other, the operation has certain unavoidable risks, and must not be undertaken except when absolutely required. In the last few years great advances have been made both in defining the indications for operation and in the methods of performing it. The field of operation has been widely extended, and the necessity for more thorough procedures has been recognised.*

In all mastoid operations the opening of the *antrum* is the first object. This cavity is really an essential part of the middle ear. It communicates by means of a large triangular opening, the *aditus*, with the upper part of the tympanic cavity or *attic*, the floor of the antrum being below the level of its opening. The attic contains the greater part of the ossicles, and is partly shut off by folds of mucous membrane from the lower division of the tympanic cavity. Thus the middle ear forms a large and very irregular cavity, divided up by its osseous configuration and by folds of mucous membrane into many more or less distinct cavities. This complicated irregularity accounts for the difficulty of eradicating an infective disease which has obtained a firm hold on it. The antrum is

constantly present even in infancy, when it is often comparatively large. In disease its size varies a great deal; it may be expanded and very large, with its walls more or less deficient, or the mastoid bone may be sclerosed, and the antrum very small and deeply situated. Its size in no way corresponds to the prominence of the mastoid process.

Its relations to the surface of the temporal bone are very important. The centre of the antrum corresponds to a point about a quarter of an inch behind the bony external auditory meatus, and on a level with its upper border. Just above the meatus is usually seen a prominent ridge on the bone, running backwards horizontally and continuous with the zygoma. The antrum lies below this ridge and the floor of the middle fossa above it. The lateral sinus lies below and behind the antrum, and is always beyond a radius of one fifth of an inch from the point above mentioned as the centre of the antrum. The canal for the facial nerve forms a prominent ridge on the floor and inner wall of the aditus, and comes into danger if the mastoid is opened too low down, or if the instruments injure the floor or inner wall of the aditus whilst opening up the attic. The horizontal semicircular canal may also be injured, as it lies on the inner wall of the antrum.

The indications for opening the antrum depend a very great deal upon whether the suppuration in the middle ear is acute or chronic. In the former case, the ear being previously healthy, acute and even very severe symptoms frequently subside spontaneously or under appropriate treatment, and the necessity for operation is very infrequent; in the latter case the conditions are exactly reversed. I propose to briefly review the course of an attack of acute suppurative otitis media, and to point out those conditions which may arise during it and necessitate operative interference.

In acute otitis the whole mucous membrane lining the tympanum is swollen and congested, the periosteum which is continuous with it is similarly affected and the Eustachian tube is blocked. Exudation, at first mucous and serous, but soon becoming

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purulent, is poured out from the inflamed surfaces, and accumulates in the tympanic cavity. The membrana tympani shares in the inflammation and becomes softened. Soon the accumulating fluid bulges it at some point, it ulcerates and gives way, and there is a discharge of blood and pus from the external auditory meatus. These conditions give rise to well-marked symptoms, acute, often intense, earache and headache, combined with a feeling of fullness and throbbing in the ear, deafness, and marked febrile disturbances. In children the symptoms are especially severe; convulsions are not rare, and, combined with sharp crying out from pain in the head, may make the affection closely resemble meningitis. The inflammation of the middle ear spreads to all the parts in direct continuity with it. Thus we find swelling and tenderness of the external auditory meatus, rendering an examination of the drum painful and difficult, with, in the severer cases, a certain amount of swelling and redness, with marked tenderness in the mastoid region and just below the auricle. Inflammation of the glands under the upper part of the sterno-mastoid is also generally met with. The severer cases are those occurring during the course of the specific fevers, scarlet fever, measles, influenza, typhoid, diphtheria, &c. After the membrane has ruptured or been incised and the discharge has gained a free outlet all the symptoms should rapidly subside, and under appropriate treatment healing should take place.

In a few cases, and usually in those who are debilitated, or in those in whom the affection has arisen during the course of one of the specific fevers, this favorable course does not ensue, or after a short interval of improvement severe symptoms again set in. This latter may be due to some obstruction to the outflow of pus, as by closure of the perforation in the drum. Such conditions should be at once sought for, and remedied by treatment similar to that at first adopted.

In some cases, however, the pus may be locked up in the antrum, or the mastoid cells and bone extensively involved from the intensity of the primary inflammation. This gives rise to severe and well-marked symptoms. The most prominent are intense earache, often deep down in the ear, and radiating to the occiput or over the whole side of the head, pain and tenderness over the mastoid region, quickly followed by swelling, redness, and

cedema. The auricle stands out from the side of the head, and also appears to be very considerably depressed. At the same time the patient is seen to be seriously ill, vomiting and giddiness are sometimes seen; there is high fever, often accompanied by rigors, or in children by convulsions. If not treated the mastoid bone may be perforated, and pus form under the periosteum of the mastoid process, or in rare cases under the upper part of the sterno-mastoid. These are the classical symptoms of an acute mastoiditis, and always indicate the necessity for immediate operation. Similarly immediate operation is required in all cases in which there are symptoms pointing to pyæmia, such as continuous high fever, repeated rigors, &c., or signs of meningitis. Meningeal symptoms are not uncommon in these cases in children, and may or may not indicate a slight amount of meningeal inflammation, but as a matter of experience they usually disappear immediately the antrum is opened.

In many of the severer cases of acute middle ear suppuration local mastoid symptoms in less degree than those above described are found. In such cases frequent hot syringing, energetic leeching, &c., should be first tried, and operation only resorted to when the symptoms continue unrelieved or progress in spite of it. Conversely we may have symptoms pointing to the retention of pus in the ear, pain, high temperature, &c., without local signs of mastoid inflammation, and in these cases also incision of the drum and other simple treatment should be tried in the first instance. For in these cases, the ear being previously healthy, extension of the affection to the brain cavity or sinuses, &c., is extremely rare. However, when it is found that simpler measures fail to give relief, and pain, fever, &c., continue, operation must be undertaken. When operation is decided upon, the best method is simply to open the antrum and establish free drainage. This is all that is needed in these cases, and there is no reason to open up the whole middle ear.

The operation is usually performed as follows:—A curved incision is made immediately behind the auricle down to the bone, the periosteum detached, and the auricle pushed well forwards so as to freely expose the external auditory meatus, which serves as the guide for opening the bone. A spot is then taken one fifth of an inch behind the meatus, and on a level with its upper border. A

circle drawn round this point at a radius of one fifth of an inch may be regarded as the field of operation. A hole is then made into the bone in this area, working inwards in a direction strictly parallel with the external auditory meatus, that is slightly forwards and upwards. A small chisel is the safest and most convenient instrument to use. Some operators prefer a gouge, but this instrument requires to be used with considerable force, and is liable to slip suddenly and penetrate deeper than one intends. A dentist's burr is safe but slow; special trephines, gimlets, &c., are very dangerous, and should never be used. One cannot see what is being done with them. The antrum having been opened, a bent probe is passed in to determine its extent, and it is well to remove the greater part of its outer wall. The ear is then well syringed out, and the mastoid wound lightly packed with strips of gauze. The dressings are changed daily, and the ear syringed. This treatment is continued until the discharge ceases, when the dressings are less frequently changed, and the wound allowed to close. Healing in such cases is usually complete in from four to eight weeks.

After such operations, the middle ear and ossicles having remained untouched, the ear may return to an almost normal condition. The perforation in the drum heals, the patency of the Eustachian tube is re-established, and the hearing power is very little impaired. In other cases, of course, just as after simple acute otitis, considerable deafness persists. In some the discharge from the ear continues although the mastoid wound heals, and finally in a very few cases the mastoid wound may not heal, or if healed may again break open and leave a sinus. The treatment of such cases will be discussed under the heading of chronic suppurative otitis.

To sum up briefly, in cases of acute middle ear suppuration we find—

1. That immediate operation is necessary when the classical signs of acute mastoid abscess are present, or—
2. When there are symptoms pointing to pyæmia, meningitis, &c.
3. That where there are present local signs of mastoiditis without much general disturbance, or symptoms of retention of pus without definite local signs, simple measures should first be tried, and operation only resorted to when these fail.

4. That operation is very rarely required, and almost only in those acute cases arising during the course of the specific fevers.

5. That simple opening of the antrum is quite sufficient, and gives, as a rule, excellent results.

Coming now to the consideration of cases of old-standing purulent otitis, we find we have quite different conditions to deal with, rendering it far more difficult to decide when and how to operate. In most cases it is not only necessary to treat any mastoid symptoms which may arise, but also some deep-seated disease which is the cause of the continuance of the otorrhœa.

Chronic suppuration is usually met with as the sequel of acute, and especially of that severe form arising in scarlet fever, measles, &c. Sometimes it is chronic from the commencement, and these cases are usually of a tubercular nature. Some of the other cases also undoubtedly become tubercular during their course. In treating these cases one must determine in the first place the causes maintaining the discharge from the ears, and the following conditions must each be considered.

1. Diseases of the nose and naso-pharynx, and especially the presence of adenoid vegetations.
2. Obstruction to the free exit of discharge from the ear, which may occur when the perforation of the drum is too small, or situated in its upper part, or blocked by polypi, &c. More rarely we may find obstruction in the external auditory meatus, due to inflammatory thickening of the soft parts, polypi, exostoses, &c.
3. A condition known as cholesteatoma.
4. The possible presence of a foreign body.
5. Caries of the ossicles.
6. Caries of the inner wall of the tympanum.
7. Deep-seated disease in the attic, aditus, or antrum, affecting either the mucous membrane or bones.

On first seeing a case of uncomplicated chronic suppurative otitis one adopts simple antiseptic remedies, such as syringing with unirritating antiseptic lotions, boracic acid, sanitas, weak perchloride or carbolic acid solutions, &c., after which the ear is carefully dried out, and antiseptic powders, such as boracic acid, a mixture of this with iodoform, &c., blown into it. This simple treatment may be varied by using instead of, or in addition to the insufflations of powder, instillations of alcohol in increasing strength as it can be

tolerated, or of such preparations as glycerine of carbolic acid, &c. Much depends upon the care with which this treatment is carried out, and if it is apparently unsuccessful it should be conducted by a skilled nurse or the doctor himself before being abandoned as useless. At the same time attention must be directed to any diseased condition of the nose and throat, and proper treatment adopted, and also to the general health, tonics, change of air, &c., being prescribed as required. Too much must not be expected from the removal of adenoids in this affection. These growths may undoubtedly cause repeated attacks of suppurative otitis, but each attack is usually mild, and subsides quickly with or without treatment. In time, if these attacks are permitted to continue, the middle ear may be permanently damaged and chronic suppuration be established, but in such cases removal of the adenoids alone will not suffice for its cure.

The more important conditions preventing the success of the above means of treatment may now be alluded to, and the main lines of treatment briefly indicated. If the perforation in the drum be too small, or badly situated, it may be enlarged in a downward direction, or a piece of the membrane excised. This means of treatment is not often successful, as the hole so made tends to close rapidly. In some cases intra-tympanic syringing may be tried. This is difficult and tedious to carry out both for patient and practitioner, and is not often of permanent benefit. Both these means, however, may be tried in selected cases.

If *polypi* or *granulations* are present, they must be removed by snaring, curetting, &c., and the tendency to recurrence kept down by the application of caustics, such as chromic acid, applied carefully on a probe. In these cases instillations of alcohol are especially useful.

Stricture of the external auditory meatus is always a serious complication in these cases. The commonest form is due to simple inflammatory induration of the soft parts, and this often yields to the above-mentioned simple treatment, aided by leeching or repeated blistering behind the ear. If a pedunculated exostosis is present it may be easily removed. In rarer cases we find a diffuse bony thickening or a fibrous stricture of the meatus, and in such cases associated with chronic

otorrhoea the mastoid operation is generally indicated as the simplest and most certain method of obtaining free drainage.

Cholesteatoma is a term applied to a soft whitish mass, consisting of epithelial cells, scales, pus, *débris*, &c., and containing crystals of cholesterine. It may fill the whole middle ear and antrum. The mass must be softened by oily applications, and then detached by a probe, &c., and scooped and syringed out. Syringing down the Eustachian tube is often very successful in these cases. Very rarely we meet with foreign bodies in the middle ear, usually splinters of wood, straw, &c., without any history of their introduction being obtained. Such cases are usually associated with polypi or granulations rapidly recurring on removal, and whilst curetting them away we may recognise or remove the foreign body. If the above means of treatment have failed to arrest the purulent discharge after careful and prolonged trial, we next proceed to removal of the membrane and ossicles. The indications for this little operation may be summed up as follows:

1. Where unmistakable caries of the ossicles is present.
2. Where there is a perforation in Shrapnel's membrane, with chronic discharge from it. This indicates disease in the attic, and although intra-tympanic treatment has been recommended and adopted with more or less success, in some cases the affection can only be thoroughly treated after free access has been obtained to the seat of the disease.
3. Where repeated slight attacks of earache with diminished secretion, combined with increased deafness and often headache, &c., occur. This also usually indicates attic disease. And—
4. Where cholesteomatous masses fill the tympanum, where polypi constantly recur after removal, and where suppuration continues in spite of other treatment.

In this operation the greater part of the membrane, the malleus, and the incus are removed, the tympanic cavity is freely but lightly curetted, and granulations, &c., scraped out. Free access is obtained to the tympanic cavity, allowing direct application of remedies and free escape for discharge. We now continue ordinary antiseptic treatment, combined with attention to the general health. This little operation is very often suc-

cessful, and especially in children and young adults. It should always be tried before proceeding to more severe measures, and should be followed by simple treatment for two, three, or even more months if improvement is taking place.

We are now in a position to recognise the circumstances in which it is necessary to operate on the mastoid antrum, and also to see the requirements of the operation when chronic disease is present.

The conditions calling for operation may be discussed under the following headings:

1. Those cases in which symptoms of acute mastoid abscess arise. All these cases require operation, and it should be performed without delay. The pus, having had for some time a free escape, is not just blocked up in the antrum, but the disease has probably spread, causing ulceration of the mucous membrane and caries of the bone. The caries is often extensive, may open up the surrounding cavities, and complications such as lateral sinus pyæmia, cerebral abscess, meningitis, &c., are very liable to follow. Hence the necessity for immediate operation in these cases. We also operate when mastoid symptoms are present in only slight degree; for depletive and expectant treatment, such as is proper when similar symptoms arise in the course of *acute* suppurative otitis, rarely does any good here, but only needlessly and dangerously postpones operation. With these cases may also be included all cases with symptoms pointing to lateral sinus pyæmia, meningitis, cerebral disease, &c., for the antrum should always be opened up first, although it forms in these cases but the preliminary step in a larger operation.

2. Cases of persistently recurring aural polypi. If after the drum and ossicles have been removed and the tympanum curetted, recurrence still occurs in spite of carefully conducted after-treatment, it plainly indicates deep-seated disease which requires to be thoroughly explored.

3. Cases of cholesteatoma involving the antrum. This is recognised when masses of *débris* have been removed from the middle ear, but the symptoms, discharge, &c., still continue. In these cases and also in cases of aural polypi it must always be borne in mind that acute mastoid symptoms may be set up by curetting the tympanum, snaring the polypi, &c. Thus these cases

must be kept under careful observation, and the surgeon must be prepared for the possible necessity of further and immediate operation.

4. In certain cases of atresia of the external meatus, as above pointed out, the radical operation is indicated as the simplest method of overcoming it.

5. Finally, the operation is necessary in all cases of chronic suppuration in which careful and prolonged treatment, aided by the above operations, has failed to cure the discharge. This failure indicates deep-seated disease, either of the bone or mucous membrane, in the attic, aditus, or antrum. Sometimes the failure of treatment is the only indication of this; at other times we may be able to detect definite roughened carious bone by means of a blunt probe. Cases are sometimes seen in which the antrum has perforated into the external meatus; sometimes we have signs that the facial canal is exposed, or that the semicircular canals are affected. When these latter signs are present we should proceed at once to operation. When there is simply incurable discharge with no other symptom, the time to operate cannot be definitely fixed. I think if the ossicles and membrane are cleared away, the tympanum curetted, and careful treatment persevered in for two or three months without marked improvement, it is high time to operate. I have left some cases longer, even for a year, but do not remember seeing one recover which was not vastly improved within three months, and from the extent of the mischief found at operation, have often been sorry I delayed so long. In these cases, the discharge having means of escape, the amount of disease found is often astonishing; the temporal bone may be extensively necrosed, the whole cochlea may come away as a sequestrum, and the dura mater and lateral sinus may be freely exposed and covered with granulations. In such cases the necessity for operation and the danger of too long delay are obvious; yet in many instances there is nothing at all, before operation, indicating this wide-spread mischief. In those cases in which, after simple opening of the mastoid antrum, a discharging sinus remains behind the ear, the radical operation should also be adopted.

Having decided to operate, we have the choice of many methods, remembering always that we have deep-seated chronic disease to deal with, and

that not only the antral but the whole tympanic cavity must be opened up in order to ensure success.

One method is to freely open the antrum, scrape out the tympanum, and then to establish free drainage, either by packing the wound, or by passing a drainage-tube round from the external meatus out at the wound, or by passing a metal tube deep into the mastoid wound. The mastoid wound is kept open many months, or until it is lined by epithelium, and a permanent fistula established behind the ear. This latter object is sometimes more quickly attained by turning a flap of skin from the mastoid region into the wound, so as to line it. This method is fairly successful, but the after-treatment is very long and tedious, and there are many objections, cosmetic and otherwise, to a permanent mastoid fistula.

The other method is a combination of many procedures, and was only introduced in 1892. It aims at laying the whole of the bony cavities, antrum, aditus, attic, tympanum, and external meatus into one large smooth-walled cavity, which is partly lined by the epithelial covered soft parts of the external meatus, and ultimately becomes lined throughout by epithelium. This is known as the radical operation, and is usually performed as follows:—A curved incision is made close round behind the ear, the periosteum separated, the edges well retracted, and the bleeding stopped. The auricle is displaced well forwards, and the soft parts lining the external meatus detached from the posterior and upper walls. Thus the bony external meatus is fully displayed, and acts as the guide when chiselling the bone. The chisel is now applied just behind the centre of the antrum, and thin layers of bone successively removed in a direction downwards and forwards, opening each time the external meatus. In this way a furrow is formed in the bone, ending anteriorly in the meatus. This is deepened until the antrum is reached, when its extent is explored with the probe, and its whole outer wall removed. This may be called the first stage of the operation. A probe is now passed down through the antrum and aditus to the tympanum, and the bridge of bone separating this probe from the external meatus is very carefully chiselled away. In so doing the upper and posterior wall of the meatus is removed, and the aditus and attic opened. Great care is

necessary when reaching the lower limit of this bridge of bone, for the facial nerve circles round the aditus, and its canal forms a ridge on its floor. This may be opened if we go at all too low, as the nerve becomes more superficial as it leaves the bone. Directly opposite us, also at the bottom of the cavity, is the horizontal semicircular canal, and this may be damaged by an accidental slip of the chisel. We must also remember that the dense bony cases normally protecting these structures may be weakened or even completely destroyed by caries. The tympanum is now freely exposed, the ossicles and drum are removed (if this has not been previously done), and the whole cavity thoroughly scraped out, care being taken not to injure the stapes or facial nerve as above mentioned. This ends the second stage, all the bony cavities being now laid into one.

Next, the soft parts lining the external meatus are cut through lengthwise opposite the cavity made in the bone, and a small transverse cut made at the outer extremity of this incision, so as to form two small flaps of soft parts. Deep stitches are passed through the outer angles of these flaps and out at the upper and lower parts of the wound respectively, so that the flaps come to be applied to the upper and lower walls of the bony cavity. The wound behind the auricle is now completely closed by interrupted sutures. The enlarged external meatus should admit easily a large finger, and the cavity is packed through this opening. A wet dressing of boracic lint is applied, and kept soaked in boracic lotion. The packing may be removed every alternate day at first, and the cavity cleansed by syringing. Afterward less frequent dressing is required. The external wound usually heals by first intention, and requires no dressing after ten days, when all bandages may be left off. The packing in the ear, however, must be continued for a month or six weeks. After that, if the case is doing well, the cavity may be daily syringed and powdered with boracic acid, and a little wool plug put in, and this must be continued until all discharge has ceased and the cavity is lined throughout with epithelium. This takes a variable time, two to four or five months, but the patient is not confined to the house. Many of my patients have been allowed up in four or five days, and have gone home in fourteen days.

The results of this operation, so far as I have been able to observe it, have been entirely satisfactory; the discharge has always ceased, and the wound being converted into a smooth dry epithelium-lined cavity no recurrence of disease is possible. At the same time the hearing power is by no means so much affected as might be supposed. In some the hearing remains quite good for conversation, and in many very deaf cases it has even been considerably improved.

To sum up, in chronic suppurative otitis operation is required in all cases in which there are severe or slight mastoid symptoms, in which there is obvious disease in the antrum or deeper parts of the tympanum, and especially if caries can be detected, when there is a dangerous form of obstruction in the external meatus, where there is a discharging mastoid sinus, and finally when there is simply a long-continued incurable purulent discharge. While also in acute suppuration the simple opening of the antrum and the establishment of free drainage is all that is necessary, in chronic cases the whole tympanic cavity must be freely exposed, and its various cavities laid into one. My chief aim in this paper has been to point out the great advantages the radical operation has over all other methods of operating in chronic purulent otitis media, and to insist on the necessity of carrying out this operation in all cases where the discharge persists after other means of treatment have failed.

CLINICAL LECTURES ON URINE.

Delivered at University College Hospital by

J. ROSE BRADFORD, M.D., F.R.S., F.R.C.P.,
Physician to University College Hospital.

IV.

GENTLEMEN,—Last time we considered the question of the recognition of blood in the urine, and there now remains the question as to how you determine what part of the urinary tract the blood has come from. Hæmorrhage from the kidney substance, as in acute Bright's, gives blood-casts, that is what tells us absolutely that the hæmorrhage is derived from the kidney substance. Blood-casts are to be seen not only in active, but also in

passive congestion, as in cases of mitral disease, so that the presence of blood-casts gives no information as to whether the hæmorrhage is from active or passive congestion, but it only gives evidence that the hæmorrhage comes from the kidney substance. In some cases of granular kidney copious hæmorrhage occurs, and it is probably derived from the pelvis of the kidney, and under these circumstances blood-casts are absent, but a considerable number of the pyriform epithelial cells characteristic of the renal pelvis and of the ureter may be found, and in this way the diagnosis that the hæmorrhage comes from the pelvis may be made. Another instance of the same thing is in purpura, where profuse renal hæmorrhage, not from the renal substance, but from the renal pelvis, may occur, and blood-casts are often conspicuous by their absence. In diseases of the bladder you rely mainly on the absence of casts, and on the fact that the blood and the urine are not uniformly mixed; and there may be other evidence, such as the presence of fragments of villous growths, as these form one of the common causes of profuse hæmorrhage from the bladder, so that the diagnosis of the seat of the hæmorrhage in cases of hæmaturia is mainly determined by the presence or absence of blood-casts, pelvic cells, renal cells, fragments of growth, and so forth.

The presence of blood-pigment in the urine, apart from blood-corpuscles, must now be considered. You have blood-pigment in the urine without blood-corpuscles in all cases in which the blood-corpuscles are disintegrated inside the blood-vessels. You will remember that in cases of urobilinuria there is an excess of urobilin in the urine, a result of the disintegration of blood-corpuscles; but urobilinuria occurs more especially if the disintegration occurs outside the blood-vessels. If the disintegration of the blood-corpuscles takes place inside the blood-vessels with the liberation of hæmoglobin in the blood-plasma, blood-pigments appear in the urine. The pigments are principally hæmoglobin and methæmoglobin, more often the latter than the former; acid hæmatin and hæmatoporphyrin may also occur, but the pigments we are really mainly concerned with are hæmoglobin and methæmoglobin. If you disintegrate the blood-corpuscle in the vessels experimentally by the introduction of water into the circulation, or by the injection of glycerine, you

get this hæmoglobinuria, and under these circumstances you get much more hæmoglobin than methæmoglobin, so that the urine is red. In disease methæmoglobin is the more common, and hence the urine is of a brownish-red or chocolate colour, and sometimes it is absolutely porter-coloured, owing to the large quantity of methæmoglobin and acid hæmatin present.

The main point is that when you break up the blood-corpuscles you have hæmoglobin itself in the plasma, and under these circumstances a portion of it is always excreted as methæmoglobin. After the experimental injection of water into the circulation you always have a certain but relatively small amount of methæmoglobin excreted in the urine. It is not definitely known where the transformation of hæmoglobin into methæmoglobin occurs; but it is certainly not in the urinary bladder, because the methæmoglobin can be detected in the urine obtained by catheterising the ureters, therefore it is either in the kidney or in the circulation; thus if hæmoglobin alone is extravasated into the blood-plasma the hæmoglobinuria is always associated with methæmoglobin, and this causes the brownish colour of the urine. This excretion of hæmoglobin and methæmoglobin is seen as the result of the action of certain poisons. One that produces it to a great extent is arseniuretted hydrogen. Several people have lost their lives in preparing arseniuretted hydrogen. At the opposite end of the scale you have a substance like chlorate of potash, which in large doses is said, in children (I do not know if it has been observed in adults), to do the same thing, *i. e.* to disintegrate the blood and cause the excretion of methæmoglobin in the urine. There is one substance—glycerine—which should be noticed, because it is perhaps of importance in this relation. If glycerine is injected into the circulation, this porter-coloured urine will be the result. In the human subject, of course, we do not inject glycerine, but glycerine is largely used for the purpose of acting as an enema, and of recent years there has been a use of it to induce labour and also to procure abortion. The point about it is that, if injected into the circulation, it infallibly produces this hæmoglobinuria. I mention this to you because a case has lately occurred where a patient had hæmoglobinuria after abortion had been procured, and died from the results of the hæmoglobinuria. We do not know that gly-

cerine had been used, but it is possible. If large quantities of glycerine were used for this purpose, and if some of it were absorbed, it is possible that it might have caused the hæmoglobinuria. Hæmoglobinuria when severe may cause complete suppression of urine. There are a number of other substances which, like pyrogalllic acid, toluene diamine, pyridin, aniline, &c., will cause this condition, but they are not very likely to be taken. Snake poison will also cause this disintegration of the blood-corpuscles, and so lead to methæmoglobinuria. There are a number of toxins in disease which will also cause it, and it is undoubtedly the fact, though it is uncommon, that it may occur in septicæmia. The common conditions (putting aside the chemical poisons, snake poison, and the toxins from various acute diseases) that cause hæmoglobinuria are two diseases, paroxysmal hæmoglobinuria and Raynaud's disease. They are neither of them very common diseases, but you will probably see cases of both of them. In paroxysmal hæmoglobinuria—you have already heard that it is one of the causes of urobilinuria—the blood-corpuscles break down with extraordinary ease. A man in this hospital suffering from this disease in the course of three days lost more than half his blood-corpuscles. This disintegration of blood-corpuscles inside the vessels occurs with great suddenness; a man may be fairly well, and yet on getting up and going out for a walk on a cold day the attack will suddenly come on, and very large quantities of blood-pigment will be passed in the urine. You understand that all that happens in these cases is that the corpuscles break up inside the blood-vessels and liberate the hæmoglobin which is excreted by the kidney in the form of hæmoglobin, methæmoglobin, and urobilin, producing the well-known porter-coloured urine. You sometimes see a similar thing in Raynaud's disease, which is a local asphyxia of the fingers, toes, and ears.

These are the principal conditions under which you get hæmoglobin and methæmoglobin in the urine. Some observers say that it may occur in man as the result of taking quinine. I have no personal knowledge of this, but it is asserted that large doses of quinine will cause it. As regards the recognition of methæmoglobinuria, you can generally form a very shrewd suspicion, simply by the porter colour or reddish-brown colour

of the urine, according to the relative proportion of hæmoglobin or methæmoglobin; but the only accurate method is, of course, the use of the spectroscope. With the microscope you do not see any blood-corpuscles, you sometimes see a granular detritus, probably the remains of blood-corpuscles, but you do not see individual corpuscles. It is also difficult to see the decolourised blood-corpuscles in the blood, and this raises the suspicion that perhaps the disintegration of the blood corpuscles does not occur within the vessels, although the stromata of the blood-corpuscles circulating in the blood stream have been described. In the urine you generally find a granular detritus, and a good many granular and hyaline casts. You must remember that this hæmoglobinuria damages the kidney very considerably, and that you may as the result of hæmoglobinuria get suppression of urine and death. It is uncommon in paroxysmal hæmoglobinuria, but it is not uncommon in the toxic hæmoglobinurias. It has been described in diphtheria. So that the excretion of the hæmoglobin and methæmoglobin by the urine is not altogether an innocuous process; it damages the kidney and shows itself by the presence of abundance of casts under the microscope, and may lead to the suppression of urine and death. You recognise these pigments by the spectroscope. The oxyhæmoglobin is recognised by its particular bands, and the methæmoglobin is recognised by its bands, this latter having two bands somewhat like oxyhæmoglobin bands, but it has a third narrow band on the red side of the D line. Hæmoglobinuria is not a condition of great clinical importance, but it is of considerable interest because of the great quantities of hæmoglobin excreted without any blood-corpuscles. I need not say much about the presence of acid hæmatin in the urine. The hæmoglobin in the presence of the acid phosphate of soda in the urine, becomes converted into acid hæmatin, and acid hæmatin is perhaps responsible for imparting the dark brown colour to some cases of paroxysmal hæmoglobinuria and of hæmaturia. Hæmatoporphyrinuria is also mainly of scientific interest, and is especially seen in some cases after the administration of sulphonol.

Now we will pass on to the consideration of the presence of bile pigments in the urine. You will remember, as regards the bile pigments, that

inasmuch as the bile contains bilirubin and biliverdin, and also urobilin, the discoloration of the urine in cases of jaundice is often due to a mixture of these pigments. In true jaundice there is not only bilirubin or biliverdin and their derivatives in the urine, but there may also be a considerable excess of urobilin. Bile pigments appear in the urine as soon as there is any material obstruction in the bile-ducts. The bile is normally secreted under a very low pressure indeed, and the presence of jaundice and of bile in the urine was considered difficult to explain, because the biliary pressure is so low that it was thought that the secretion would cease before the pressure in the ducts was sufficient to cause the bile to pass back into the tissues. In obstruction of the bile-ducts, however, the bile gets into the circulation through the lymphatics, and it gets into the blood-stream, not through the hepatic veins to any appreciable extent, but through the thoracic duct. As soon as the bile gets into the circulation it is excreted by the kidney. The kidney picks out the bile pigments and excretes them in the same way that peptones are excreted. Not only is that the case, but the kidney is, so to speak, such a delicate organ, with such a selective affinity for bile pigments, that you may have the presence of bile in the urine when the patient is not obviously jaundiced. This, however, is most apt to occur at the commencement of an attack of jaundice. Bile appears in the urine in cases of obstructive jaundice very frequently previously to the discoloration of the skin.

On the other hand, cases are sometimes seen where, after long-continued obstruction, the skin remains yellow, but the urine is not discoloured. It is customary still to describe two varieties of jaundice, obstructive and non-obstructive, and I am afraid I must repeat what I said before, but it is very important to have clear ideas. The word jaundice is used in two senses: it is used by some people as simply meaning a yellow patient with yellow conjunctivæ, apart from what is making him yellow; it is used by others for a patient who is yellow from the presence of bile pigments in the blood. There is very little doubt that you never get true jaundice,—that is to say, a patient yellow from the presence of bile-pigments, unless the bile-ducts are obstructed, so that there is no non-obstructive jaundice. The cases of so-called

non-obstructive jaundice, *i. e.* yellow patients with yellow conjunctivæ, and in which post-mortem the bile-ducts are patent, admit of several explanations. In the first place, a considerable number of these patients are jaundiced because the obstruction is in the capillary ducts. Phosphorus poisoning, which is not seen so very frequently as it was ten years ago, is an instance of this. Phosphorus poisoning is a condition in which the patient becomes intensely jaundiced, and in which post mortem the bile-ducts are apparently patent, and this used to be quoted as an instance of non-obstructive jaundice; the capillary ducts are blocked owing to a catarrh of the fine ducts, and the pouring out of a quantity of sticky mucus; although the large ducts are patent and not distended, the patient is really suffering from obstructive jaundice. That explains one great group of cases of non-obstructive jaundice. The others are not really cases of true jaundice at all, they are cases of urobilinuria, and, as we saw last time, the colour of the urine and the colour of the patient suffering from urobilinuria is somewhat similar to the colour of the patient suffering from true jaundice, so that the unequivocal sign of bile pigment in the urine means that there is obstruction in the bile-ducts, but it does not mean that you will see the obstruction in the post-mortem room, it may lie in the capillary ducts. It is very doubtful whether bile pigment is ever formed simply as a result of decomposition of extravasated hæmoglobin; and I think most, if not all cases of so-called non-obstructive jaundice admit of the explanation just given. Bile also contains bile salts, and it is a remarkable fact that in cases of complete jaundice you do not get any appreciable quantity of bile salts in the urine. It is difficult to detect bile salts in the urine in anything but traces; there are traces present, but you cannot detect them by examining the crude urine, you must use special methods, whereas bile pigments are detected with ease. You are all familiar with the tests for bile pigments; the best test is to add fuming nitric acid to the urine in a dish, not in a test-tube. The characteristic reaction is not so much the play of colours as the fact that the play of colours commences with a green colour. Skatol and indol compounds may give a play of colour with oxidising agents, but they do not give the well-known green colour dependent on

the formation of biliverdin, therefore if you do the test, do it on a plate, and lay stress on the initial green; you will not go far wrong if you do that. You may use the iodine test if you like, the shaking up the urine with a solution of iodine giving you a green colour, it is the same thing, an oxidising agent producing biliverdin. These tests are so good, and liable to so few fallacies, that it is not worth while going to the other tests, but a useful test is the sulphur test, *i. e.* the sinking of a fragment of sulphur in a fluid containing bile. If you want to determine the presence of bile salts, which you really never do want to do clinically, you must evaporate the urine and extract with alcohol, and you must examine the alcoholic extracts for bile salts with Pettenkofer's reaction. This test will not succeed in the majority of cases if the ordinary urine is used, owing to the fact that it contains such a small quantity of the bile salts. Dr. Brunton pointed out that if you have a case of intense jaundice you can get Pettenkofer's reaction by shaking the urine violently, and the characteristic colour will be seen in the froth. As regards the colour of the urine, that which contains bile is always greenish at the top. It may be almost any colour in the substance of the urine; it may be red, brown, or it may be almost black. It depends entirely on the amount of bile present, but it is always greenish on the top, and none of the skatol, indol, and urobilin pigments cause the urine to be green on the top. There are clinically three varieties of jaundice: there is first of all the well-known class of jaundice which our patients call the "yellow" jaundice; then there is green jaundice, and finally there is black jaundice, which is a rarer condition. The question as to the colour of the jaundiced patient is entirely a question of the duration of the obstruction. If this lasts for several months the patient becomes green, and a green tinge is often seen after as short a period as six weeks or two months; after about a year the patient becomes very dark. The green colour is obviously due to the pigment becoming converted into biliverdin, but in black jaundice the nature of the pigment is not so definitely known. There was a case in this hospital some years ago so black from jaundice that the man was almost indistinguishable from one of the darker races. That patient was suffering from cancer of the pancreas with complete

obstruction of the bile-ducts, and the jaundice had lasted for more than a year. On the other hand, I have seen a patient jaundiced continuously for six years, but the colour of the skin never went beyond the green stage.

ADENOIDS.

BY

CAMPBELL WILLIAMS, F.R.C.S.

ANYONE looking through the list of minor operations at a children's hospital must be struck by the frequency of the entry "Adenoids," either alone or in conjunction with "Tonsils."

Meyer of Copenhagen was the first to draw attention to overgrowth of the post-nasal adenoid tissue. Prior to this discovery the tonsils were looked upon as the sole cause of troubles which are now known to be due, either wholly or partly, to the presence of adenoids. When the tonsils are enlarged it is almost a certainty that adenoid vegetations co-exist to a greater or lesser degree; but, on the other hand, adenoids may be present in considerable amount when the tonsils are practically of normal size.

The importance they bear to the general health and welfare of the child is so fully recognised that it may account for the great number of cases that come to operation. Any observant man, in his daily walks, can pick out children whose faces too plainly tell of the existence of some obstruction to free respiration. The pinched or flattened nose, often fretted at the nasal apertures from continuous discharge, the open mouth, the vacant staring expression of the eyes, speak for themselves. In many an ailing child, who cannot be said to be definitely ill, one can trace its impaired health to enlarged tonsils and adenoids. As regards the latter, it has been most pertinently said that it, is not so much the quantity present as the effects produced that call for operative interference. Adenoid vegetations may be met with at all ages, from the first month of life onwards. Cases usually come under the surgeon's care when they are from two to seven years old. The sym-

ptoms which most frequently draw attention to their existence are—

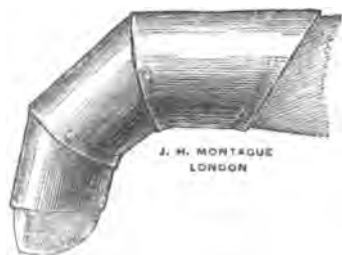
1. Snoring.
2. Imperfect speech or nasal speech.
3. Chronic nasal catarrh.
4. Aural discharge.
5. Deafness.
6. Open mouth.

If the amount of obstruction to respiration is great, and is not attended to until about the seventh or eighth year of life, it will be noticed that the development of chest is below the normal standard. The impaired inspiratory power has not allowed of full expansion of the lungs, and the tissues of the body in general suffer through deficient aëration of the blood. The patient is often pale, flabby, and weakly. The breathlessness produced by exercise makes them disinclined to run or play as other children do, and consequently their muscular system is ill-developed. The term *listless* is most applicable to this class of case. Again, owing to nasal obstruction, having to use their mouths not only for eating but for breathing purposes as well, they bolt their food. Their main desire seems to be to get through the meal as quickly as possible. The snuffling, "yaffling" noise that they make whilst feeding is very typical. The results of obstruction to the Eustachian tubes is a factor of serious import in these cases. From it they may get stenosis, or, from catarrh spreading up to the middle ear, a chronic aural discharge which does not subside until the adenoids have been removed. The impairment of the power of hearing may vary from loss of acuity up to absolute deafness. If one studies the school report of an "adenoid boy," it is usually of the "backward" type, and the entry "inattentive" stands out most prominently. The boy may not be deaf in the ordinary acceptance of the term, but owing to loss of auditory acuity he has to continually concentrate his attention in order to "take in" what is going on. In more advanced cases they are stupid, as they cannot hear properly what their teachers say, and consequently fail to understand. They not only look, but are vacant. Partly from want of use and training their mental capabilities for absorption or retention of their lessons are of a low order. Deafness at first may be intermittent, coming on whenever the child gets a cold in the head, and relapsing to the "impaired stage" as the catarrh

and swelling subsides. But if the cause of this condition is not rectified *by removal*, there is a distinct danger of it becoming permanent later on.

The sleep of a "tonsil adenoid" child is characterised by deep snoring, and it is not uncommon for it to start up from its slumbers as if from a sense of suffocation. Imperfect oxygenation of the blood acts as a predisposing if not an actual cause in the production of *nocturnal incontinence*. This is probably due to the action on the lumbar centres, which govern micturition, of the insufficiently aerated blood. In cases that have persisted after circumcision and treatment by drugs I have broken the habit by removing the tonsils and adenoids which were present. Their removal also improves many children who stammer, as clearing out the nasopharynx not only allows them to take greater and deeper inspirations, but creates an upper resonance chamber if they should have to "sing their speech." It is worthy of note how the general health improves after operation, the thin, anæmic child becoming fat and rosy. Chest measurement rapidly increases, and violent exercise can be indulged in with pleasure and comfort.

When enlarged tonsils are present, adenoids should always be suspected. It is of little use to remove the former and leave the latter. By passing the finger behind the soft palate their presence or absence can be determined. The examination is made almost before the patient is aware of what is going to be done. The amount and consistency varies. The posterior nares may be completely occluded by large pedunculated masses, or they may be small and sessile. Sometimes they may be found on the upper surface of the velum palati.



As a rule they are soft and bleed readily, but when they have existed for any length of time they are apt to become fibroid. Special metal guards are made for the protection against biting of the examining fingers, but a couple of turns of lint

around the digit suffices, more particularly so if it is introduced by the side of the temporary molars, where there is no cutting edge to the teeth.

Should an operation be determined upon, and I will here repeat that the effects produced by a small amount of adenoid vegetations often warrant one, the choice of an anæsthetic has to be considered. There is great difference of opinion as to which is the best. Some surgeons prefer gas, others ether, whilst many adhere to the A.C.E. mixture. But whichever is determined upon, specific and imperative direction should be given as to the feeding of the child on the day of operation. If one elects to perform it in the morning, all that is necessary is to see that the patient has no breakfast. If the afternoon is chosen, say 2 o'clock, the child should have a good breakfast, *without meat*, at 9 a.m., and if it is thought desirable, a little beef tea at 11. After this nothing should be allowed to pass the lips. A good deal of the trouble that arises in these cases, during the administration of anæsthetics, is due to the presence of food in the stomach. Although the parents may assure one that the instructions as to diet have been implicitly obeyed, it is not an uncommon occurrence to find that the child has managed to get hold of food somehow or another, as is proved by the undigested character of the vomit. Many "adenoid" children take anæsthetics badly, presumably through impeded respiratory powers, and it behoves one to be watchful and prepared for emergencies. I make it a rule to always have the body stripped to the waist in case of accidents, so that one can clap on a hot towel over the heart as a cardiac stimulant, or inject ether over the region should it be required. Artificial respiration is likewise more easily performed, and should be promptly applied if needed, the tongue being drawn forwards with forceps. Personally I prefer the A.C.E. mixture, but they should not be too deeply under its influence. The idea is to retain the sensitiveness of the glottis, and thus prevent the entrance of blood or foreign bodies into the windpipe. The usual preliminary examination of the heart does not, in my experience, give a clue as to whether one *may* or *may not* expect a *syncopal attack*. This is not an uncommon occurrence, and it may happen at any period during administration from the first few whiffs onwards.

The best position for the child to be placed in for operation has been the subject of considerable discussion. Some surgeons like the patient *to sit* on the knee of an assistant, who restrains the arms and keeps the head bent slightly forwards. It is claimed for this attitude that one can *see* better to remove the tonsils, and that the hæmorrhage that results from erasing the adenoids has a free outlet by the nose and mouth, and does not tend to gravitate towards the larynx. Others advocate that the patient should lie on the side, with the head inclined towards the chest. The position that I like best is the dorsal. The child is placed supine on a table of convenient height, so that the head just reaches the end. A slop-pail with a large mackintosh-sheet beneath it is put below to catch the blood. The anæsthetic is given, and the tonsils



excised, if necessary, in this position. As soon as this has been done the patient's head is pulled on so that it hangs downwards over the end of the table. The adenoids are now removed, preferably with a Gottstein's knife. The child is then rolled over to one side, so that the mouth looks

towards the pail, and the finger-nail is used to complete the clearing out. The oral and nasopharyngeal cavities are mopped out with pads of boracic lint. As soon as the bleeding shows signs of cessation the child is drawn up *flat* on to the table, and the head and body turned slightly to one side. Needless to say that the amount of hæmorrhage varies. The soft adenoids bleed much more freely than the fibroid, and the quantity of blood lost is likewise influenced by the plethoric condition of the patient, the amount of venous congestion present, and *the time that the operator takes over the case*. But it is surprising how quickly the bleeding ceases, more particularly so when the head is slightly raised and the gag is eased; but it may occasionally be necessary to apply pressure by means of pads to attain this end.

With the head hanging downwards and backwards one has great command of the knife, and the blood that does not escape by the nose passes down the œsophagus to the stomach, and is subsequently vomited. In the supine position a good view of the tonsils can be obtained by pulling the tongue forwards by means of forceps. To expedite their removal it is advisable to have two gags. Say that the right tonsil is to be excised first. A gag is put in on the *left* side of the mouth, so as not to be in the way of the guillotine; and as soon as the tonsil is out the second gag is placed on the right side, and the original one removed. By this device it is impossible for the child to close the jaws, and no time is lost thereby.

Löwenberg's forceps, Meyer's ringed knife, and the artificial nail have practically been discarded for Gottstein's instrument. The last is most effective and rapid in its action, but care should be taken not to lateralise the blade, for fear of injuring the ends of the Eustachian tubes, and to restrict its use to the posterior plane of the naso-pharynx. A few well-directed strokes suffice to detach the greater portion of the vegetations. The finger-nail is used to complete the removal as well as to explore the upper surface of the soft palate and the fossa of Rosenmüller. A satisfactory operation should leave a practically smooth surface to the touch. There is often slight oozing from the nares after erosion, which may persist for some minutes, so that it is advisable not to allow the parents to see the child just immediately, as it is apt to upset nerves that are already in a state of tension. One

should also warn them not to be frightened if, after one has left the house, the child should be sick and throw up swallowed blood.

The routine after-treatment consists in keeping the patient in bed until the next day, and confining it to the room for the following three or four days. In cold weather, a week to ten days is a safer course. I do not order the use of nasal douches or sprays. Young children resent their employment, and struggle so much as to render their application difficult. Moreover, experience shows that they do perfectly well without them. In older children, with power of understanding, whose tonsils have been excised, I order the following gargle :

R Glycerini Carbol.	℥ xv
Glycerini Boracis	ʒj
Aq.	ad ʒj

Use 4 times daily.

There is usually a good deal of upset after the anæsthetic and operation, and the first night's rest is fitful and disturbed. The tongue becomes furred from gastric disturbance, and may demand a dose of grey powder with rhubarb and soda as a corrective measure. Most children are improved by taking Vinum Ferri ʒj three times daily for a fortnight after operation.

Though it may be difficult to convince a fond mother that her child is in no danger of starvation; it is best to withhold all food for some hours after the patient has "come to," for it only causes sickness when given too early. Later on slops may be taken. Often about the second day the child develops an appetite that formerly was quite foreign to its being, and eats regardless of faucial discomfort.

The snoring, though it may be modified, does not disappear immediately after erosion. This is presumably owing to the swelling that results from the usage of the parts; but when this subsides, as it does in about a week's time, the sleep is quiet and peaceful. The "open-mouth aspect" may persist for a while, but this is due to the habit acquired rather than to any respiratory obstruction. As possible complications that may arise after operation, one must bear in mind septic meningitis and pneumonia, and also that suppuration of the middle ear may supervene. So far it has been the good fortune in my cases to escape with only slight earache; but the old adage of the "pitcher and the well" must not be forgotten.

NOTES.

The use of Antistreptococcic Serum in the Treatment of various forms of Septicæmia.

—The very interesting studies which have been made during the last few years in regard to immunity, and our ability to produce it in animals and in human beings, has resulted, first and foremost, in the employment of diphtheria antitoxin for the protection of persons who are infected by the Klebs-Loeffler bacillus, and the extraordinary results which have been obtained by the use of this serum in the treatment of this disease have placed its use upon as firm a basis as that upon which any therapeutic measure can rest.

Naturally experimenters and clinicians have gone further than this, and have attempted to use other serums prepared in ways identical with or only slightly different from the methods employed in the preparation of diphtheria antitoxin, for the protection of individuals suffering from an infection with other malignant germs than those of diphtheria. One of the most noteworthy attempts of this character has been the employment of the so-called antistreptococcic serum, or the serum obtained from animals supposed to be rendered immune to the streptococcus, in persons who are suffering from septicæmia following injuries, surgical operations, or parturition. While the scientific information upon which this treatment is based amply justifies the employment of antistreptococcic serum and other serums destined for similar purposes, it is an unfortunate fact that the results which have been obtained by its employment so far have not been such as to give us the encouragement which the early studies with regard to diphtheria antitoxin gave us. Medical literature, particularly in England and in Europe, has teemed during the last few months with isolated reports of cases of septicæmia treated by this means, and some clinicians have even gone further and employed this serum in the treatment of such a disease as scarlet fever, in which, in addition to a specific infection, it seems probable that grave streptococcic infection also occurs. It is worthy of note, too, that in the treatment of scarlet fever results have been obtained which would certainly justify the experimenters in continuing the trial of this substance, although they have hardly been

sufficiently numerous or so universally successful as to justify the average American physician in following in their pathway. Perhaps the most notable contribution which has been made in American medical literature to the study of this question is a series of papers recently communicated to the Section on Gynecology of the College of Physicians of Philadelphia by Hirst, Norris, Shoemaker, Davis, Baldy and Shober. These papers combined are so exhaustive that it is not possible for us to give more than a summary of the views of their individual authors. Dr. Hirst, from his experience in the employment of antistreptococcic serum in puerperal sepsis, believes we are not in a position to pronounce any judgment on this new treatment; that we should not be so prejudiced against it as not to give it a thorough clinical test, and on the other hand should not be too enthusiastic concerning it. As he well points out, the following forcible objections can be urged against this method of treatment: First, the well tried, older plans of treatment will result in the cure of about four-fifths of the cases. If, therefore, the serum is employed along with other suitable treatment, four out of five cases will be cured in all probability, and some of these cures may be erroneously attributed to the serum. Next, he points out the difficulty of obtaining a thoroughly reliable preparation, which difficulty has been largely obviated within the last few weeks by the placing upon the market of a reliable preparation. Third, he believes that the use of this remedy must be always more or less empirical, for while it is true that most cases of puerperal infection are due to streptococci, there are others which suffer from a mixed infection or in which the streptococcus is not present at all. Again, the treatment is not entirely free from risk, for several French observers have reported deaths in which the result was apparently unmistakably due to the serum. Again, its use may cause the physician to be careless in the employment of other necessary remedial measures. And, finally, we know nothing as to the real method by which the serum does good. Norris concludes the report of a case in which he employed the serum by stating that very strong clinical evidence of streptococcic infection should be had before resorting to the employment of antistreptococcic serum in the treatment of puerperal sepsis.

Shoemaker's case, one of septicæmia occurring after abortion, was treated in this manner, but the patient nevertheless died. Davis expressed the belief that this method of treatment must be employed early, in connection with stimulating agents, and we may then hope for a good result in a small number of cases if they are seen promptly; while Baldy seemed to think that the death of his patient resulted from the administration of the serum. He states that while it is possible that death occurred by coincidence after the administration of this treatment, he will nevertheless in future use the serum, if at all, with the utmost caution and distrust.

The contribution to this symposium made by Shober consisted in a series of twenty-one cases collected from the *Lancet* and *British Medical Journal* during 1896. Shober concludes that to obtain the best results it is of prime importance to obtain reliable serum, that the case should be one of unmixed infection, that the injections ought to be commenced upon the onset of symptoms, and, finally, that it is highly important to employ intra-uterine and vaginal treatment in connection with the serum treatment.—*Therapeutic Gazette*, August 16th, 1897.

Transcendental Surgery. Efforts to secure Absolute Asepsis, Gloves and Mouth Screens.

—Prof. J. Mikulicz contributed a candid and most interesting article to the 'Deutsche Med. Woch.' of June 24th, on the open secret that the results of asepsis are scarcely any improvement over anti-sepsis. The most scrupulous care by conscientious surgeons and assistants is insufficient to guarantee absolute asepsis as an actual fact. He has recently adopted in his clinic two innovations, which he considers long strides towards the attainment of this ideal: gloves for the operator and assistants, and a covering over the mouth of each person in the room. He finds from a long series of tests that it is impossible to render the hands perfectly aseptic. The manipulations required of the surgeon's fingers bring to the surface germs deeply ensconced in crevices impossible to reach by the most vigorous disinfection. He has therefore commenced to wear gloves at his work—not the rubber gloves recommended by Manteuffel and others, nor the long silk gloves advocated by Perthes in the 'Cbl. f. Chir.' of July 3rd, but the

cheap gloves sold as "fine servants' gloves," waiters' gloves, which he buys in Breslau for 65 cents a dozen. They are linen or cotton, and can be washed and boiled, and used over and over again. He first disinfects his hands as carefully as possible with the alcohol-sublimate method, and then draws on the gloves. They do not interfere with his operation, and even allow a firmer grasp of the threads and tissues. If the operation is short and aseptic, one pair of gloves is sufficient; but if not, he changes for a fresh pair two or three times, at the different steps of the operation. His assistants also wear the gloves, and change at the same time. If absolutely necessary to use the bare finger, he removes the glove for the purpose. Of course, he does not wear them when opening up an infected focus. The constant agitation in regard to improved methods of asepsis and the catgut question, drainage, &c., proves that surgeons are not fully satisfied with the present methods at the best, and Mikulicz's suggestions have already been adopted by others. Küster, for instance, has commenced to wear the gloves, and announces that he is pleased with them in every respect. Mikulicz has also found that germs are disseminated in the air from the mouth in speaking or coughing, floating on tiny bubbles of moisture. As moist germs are much more dangerous than dry ones, to reduce this evil to the minimum he limits the number of persons present at an "asepsis operation" to the smallest number possible, not even admitting more than six to ten students at most, and all present wear a sterilised piece of mull over their mouth, fastened to their sterilised cap; it can also enclose the beard if there is one. They soon learn to breathe through it as comfortably as a lady through her veil. Gestures take the place of words as much as possible. One eminent bacteriologist considers that a surgeon with a cough or tendency to sneeze, has no right to attempt an "asepsis operation." The germs that may linger on the patient's skin after disinfection are not usually so virulent as those on a surgeon's fingers, but still Mikulicz considers that drainage is frequently a source of infection, as the germs of the surrounding region find their way into the wound along the drain, especially if near the anus, &c. He never attempts an "asepsis operation" in the clinical amphitheatre before a crowd, but floods everything there with antiseptics.

He recommends all surgeons to use antiseptics in operating at the residence, as only a perfectly aseptic room in specially equipped institutions will insure success. König acknowledged at the recent German Congress of Surgery that he had learned from experience that suppuration of the knee-joint did not occur after patellar suture if the finger did not come into actual contact with the tissues, which is an argument in favour of gloves; even Kocher's painstaking technique has failed to prevent suppuration in 8.7 per cent. of his radical hernial operations.—*Journal of the American Medical Association*.

On the Importance of the Irrigation of the Nose.—'Petersburg Med. Woch.,' 1897, No. 24. Borgengrün, the author, gives the following conclusions:—1. In very young children irrigation of the nose should only be done by an experienced hand. 2. The liquid must not be injected by force. At least half an hour after the injection the patient is not allowed to blow his nose hard. 3. During the irrigation there must not be any phonation or swallowing; as soon as this happens the injection must be left off. Also, when the patient feels any sensation in the ear, injection must cease. 4. There must always be intervals after every five to ten cubic centimetres injection. 5. The liquid must not be too warm or too cold. Solutions of alum or carbolic acid must not be used. At the end the author mentions the different instruments for irrigation of the nose.—R. SACHS. *Journal of Laryngology, Rhinology, and Otology*, September, 1897.

A New Sign of Phrenic Neuralgia.—Before the Paris Société Médicale des Hôpitaux, at its meeting of July 30th ('Journal des Praticiens,' August 7th), M. Jousset insisted upon the existence of a constant painful point situated precisely in the median line of the sternum, at the level of the fourth or fifth chondro-sternal articulation. It should not be confounded, he said, with the diffuse retro-sternal pain observed by Peter in chronic affections of the aorta. The point was of importance, absolutely decisive, in cases in which one was in doubt whether to refer an epigastric pain to the diaphragm, the gall-bladder, the stomach, the abdominal wall, or the intercostal nerves.—*New York Med. Journ.*, August 28, 1897.

THE CLINICAL JOURNAL.

WEDNESDAY, SEPTEMBER 15, 1897

SOME CASES OF INTEREST IN MAMMARY DISEASE,

With Remarks.

By A. MARMADUKE SHEILD, M.B., F.R.C.S.

PART I.

AMONG the cases of carcinoma of the mamma of great interest and importance which have come under my notice in the last six months the following may especially be quoted:

A very stout, ponderous woman, æt. 60, came under my care by the advice of Dr. Shann, of Lowestoft, with a large carcinomatous tumour of the left mamma. The patient was a typically bad subject for an operation. In addition to obesity she was bronchitic, and the cardiac sounds were feeble. There were enlarged venules on the cheeks, and a trace of sugar in the urine. The local conditions were still more unfavorable. The mamma was of huge size, and converted into a mass of cancer. The skin was extensively implicated, being red, hot, and thickened, with an erythematous blush round. The nipple was puckered and retracted. The axilla contained a mass of enlarged glands. At 'Consultations' removal was nevertheless advised on account of the fungation which would shortly occur. Local relief was alone anticipated, and anything like long immunity was not to be hoped for. The disease gave the patient little pain, and so far as could be ascertained, the cancer had been advancing for about twelve months. The operation was performed in the following way: two days were spent in the purification of the skin, especially of the axilla. The incisions were carried far and wide of the diseased skin, and reached from the coracoid process to the sternum. The whole pectoral muscle fascia, axillary glands, and mamma were removed in one piece. Several glands were found on removal of the pectoral which would probably have escaped detection had minor measures been adopted. The resulting wound was of huge size; it was drained posteriorly and brought together as much as possible. Owing to

the free removal of skin an area the size of a small dinner plate was left bare. In this situation I raised a series of tongue-shaped flaps from the edges of the wound, turned them inwards, and sewed them down upon the denuded surface. The ordinary antiseptics were used in dressings. All went well until the second week, when the patient showed signs of mental failure, as talking nonsense, refusing food, and sinking into the bed. This was met by free stimulation and getting the patient into a chair. The mental symptoms passed off in about three days. Iodoform was being used to the wound at the time. By the end of eight weeks this huge wound was soundly healed, and the condition of the patient very good. There was no sign of local return, and the condition of fungation of a huge mass of cancer was quite obviated. This patient remains well four months after operation, with no trace of return of disease, a result which surprises me greatly, as I could scarcely imagine it were possible all could be removed.

Remarks.—This case illustrates what can be done in mammary surgery by the aid of careful antiseptics, and that it is justifiable to operate on a case even if the local and general conditions are highly unfavorable. The involvement of the integument in this case was of so extensive a nature that only a very wide and bold course of incision rendered operation at all feasible. The case also illustrates that operations in apparently very bad cases of mammary carcinoma are sometimes followed by excellent results. Mental disturbances after mammary operations have several times been observed, and I have notes of other cases. Some have died acutely maniacal—they have usually been intemperate. It is noteworthy that in the practice of some surgeons, mania was associated with extensive use of iodoform. The cases differ very widely, and experts in lunacy could alone give reliable prognosis. The treatment resolves itself into careful nursing, and the most careful dressing of the wound for such patients are apt to displace their dressings, and sepsis is added to the already present troubles. The pulse will be the guide for

stimulants, and for hypnotics; enemata of bromides are often advantageous. The stronger hypnotics, as hyoscine or morphia, must be used with the extremes of caution.

The next case is illustrative of a most important class, where cancer is developed in an outlying lobule of mammary gland. Such cases are stated by many authorities to be really cancers growing

axilla, yet the nipple was progressively drawn towards the growth at a considerable distance from it. In this case, as may be expected, the axillary glands were much implicated. When the cancer is developed quite to the sternal side of the mamma, the axillary glands may long escape, as in the following instance.

On June 21st I operated on a strong, well-

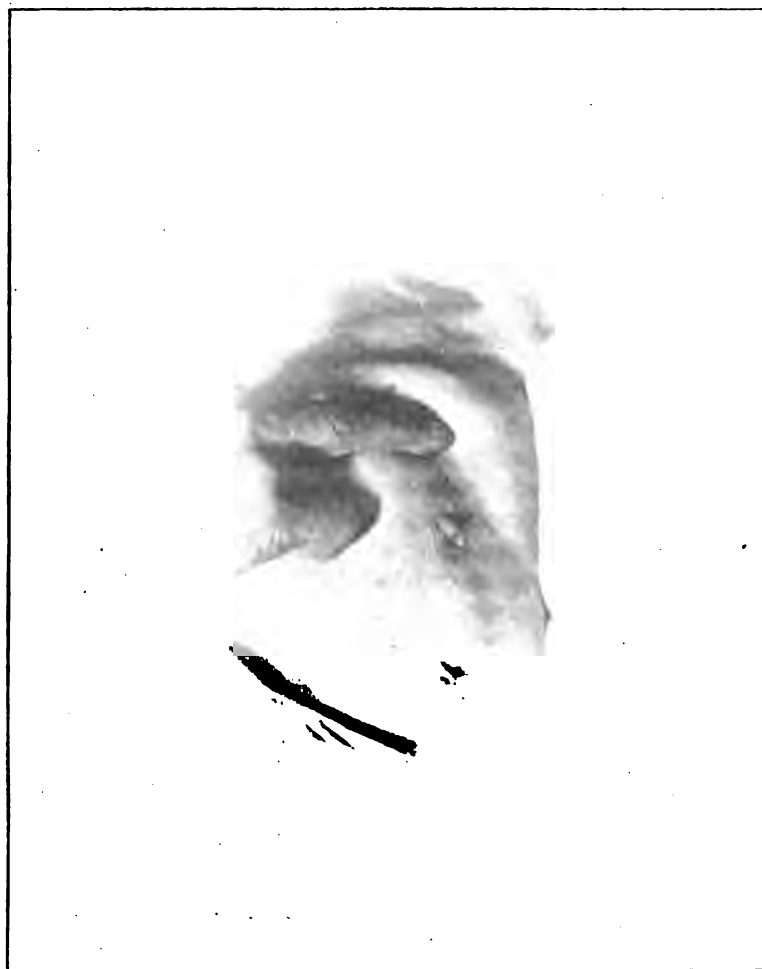


Fig. 1.—Carcinoma of a peripheral lobule.

in accessory mammae. As a matter of fact most of these little islets of mammary tissue are connected with the main gland by fine strings or processes of glandular structure. A common situation for peripheral carcinoma is at the junction of the mamma with the thorax. The serious disease is merely marked by an insignificant dimple in the skin (Fig. 1). A case has recently been under treatment where the carcinoma developed high in the

nourished lady aged about 45, who had had children and nourished them on the affected side. The history was that a slight contraction had been noticed about the mamma for twelve months, but with trifling pain or inconvenience. The nipple of the left mamma was deeply retracted, and close to the margin of the sternum was a faint dimple, which might seem quite disconnected with the mamma. The rest of the breast gave the sensation of mas-

titis, and the only sign to be felt with the "flat hand" was an abiding hardness beneath the dimple above spoken of. At the operation the mamma, sternal portion of pectoral, with the fascia and axillary lymphatics, were removed in one piece. There was a nodule of carcinoma the size of a large hazel nut deeply situated in the left fourth inter-space, close to the sternum. It had infiltrated and involved the pectoral muscle and the fascia covering the internal intercostal, and on its removal the greatest care had to be taken to avoid opening the pleural cavity. Not a trace of disease was found in the axillary glands. The rest of the mamma, however, was far from healthy, and indurated cords of cancerous tissue radiated from the sternal process outwards. The wound rapidly healed.

This case is of interest as exemplifying that carcinoma even of twelve months' duration, when situated at the sternal side of the mamma, need not of necessity affect the axillary glands. It also exemplifies how deep and serious the implication of the chest-wall becomes in these apparently insignificant cases. The attention of the profession has hardly enough been drawn to peripheral carcinoma. It is hard to realise that a mere dimple in the skin, with an ill-defined hardness beneath it, is of grave import. Definite tumour formation is not appreciable, as is the case when carcinoma forms in the locality of the nipple. The question of removing the whole mamma must often arise in these peripheral cases. Instances are not wanting to exemplify the practice of merely excising the outlying disease and leaving the mamma. The breast substance is, however, seldom uncomplicated, and far the wiser practice is to perform the widest possible removal. Owing to the early implication of the chest-walls, cases of peripheral carcinoma cannot be too soon treated. They are really among the most urgent cases for immediate operation.

The next case is a remarkable instance of a very rare affection, the so-called Paget's disease of the nipple, a malignant dermatitis. In the cases of carcinoma of the mamma occurring at St. George's in thirty-five years, I have been surprised to find that barely 1 per cent. of the cases of mammary cancer were preceded by this condition.

A woman æt. 42, married, and the mother of children, came to the out-patient department at St. George's Hospital in 1896. The history she

gave was as follows:—Eight years before a mammary abscess burst near the right nipple. The sinus never healed, and the present disease originated round the orifice, and not round the nipple, of this she was certain. It had gradually extended with increasing "itching and burning." Now the pain was severe, disturbing her rest, and seriously interfering with her rest and health. On inspection



Fig. 2.—Malignant dermatitis of the integument over the entire mamma.

a very remarkable condition was apparent (Fig. 2). There was a large oval patch of disease occupying the skin of the entire right mammary region, reaching to the apex of the axilla above, the sternum in the mid line, and the latissimus dorsi behind. The patch measured fourteen inches vertically by twelve inches horizontally. It was vivid crimson in colour, covered with islets or patches of white epithelial scales, moist, and exuding abundantly a sticky glutinous secretion of alkaline reaction and musty offensive odour. The margins were as strictly limited as though the disease had been artificially produced. There was definite parchment-like thickening of the tissues, easily appreciated on pinching up a portion. At the orifice of the old sinus was a fungating carcinomatous nodule the size of a walnut. No glandular affection in the axilla could be recognised. The case was shown at the Dermatological Society of London, when most of the members recognised

the disease, and remarked upon its unusual extent. It may be mentioned that "scrapings" of the surface showed abundantly those forms of cells, and cell inclusions, termed by some porosperms.

In April, 1897, this patient's sufferings were so great that she entered the hospital and begged for any treatment likely to afford relief. The only application that gave any local comfort was a 5 per cent ichthyol ointment. The disease had seriously increased in dimensions, and it was not without misgivings at the magnitude of the task that I undertook its removal. The operation consisted in carrying an incision round the whole margin of the affected skin, a quarter of an inch from the red border. The subjacent mamma and pectoral muscle were removed in one portion with the skin. There was an indurated and enlarged gland beneath the pectoral, which was likewise removed. The skin was removed freely with the subjacent fascia, so that the intercostal muscles were exposed. No attempt was made to unite the margins of the enormous surface thus left bare and raw, but a number of skin flaps, varying in length from three to six inches, and broader at their bases than their sides, were raised from the margins and sewn downwards upon the raw surface (Fig. 3). The wound was washed daily with warm perchloride lotion (1 in 8000), and dressed with a large piece of green protective and cyanide gauze. The flaps were adherent, and epithelial growth advanced from many points, coalescing and advancing. After six weeks some more flaps were raised, and placed upon granulating surfaces. To make a long story short, this extensive surface was covered by a sound cicatrix in about two months. A microscopical examination by Dr. Rolleston and Mr. Hunt disclosed very curious features. The disease of the skin and the structure of the nodule was epitheliomatous, large spheroidal cells and columnar masses of ingrowing infective epithelium being very evident. The mamma itself was flattened and atrophic, and exhibited no signs of cancerous disease. The infected axillary gland contained spheroidal-celled carcinoma. The present condition of the patient leaves nothing to be desired. She has lost all pain, and greatly improved in health. The large scar, traversed by bands of healthy skin, shows no signs of "breaking-down" or return of disease.

This unusual case illustrates several features of

a rare and curious malady. Its main peculiarities are its extent, and the fact that the mamma beneath was quite free from carcinomatous change, though the skin affection had lasted for eight years. The diagnostic features of vivid red rawness, thickening, and defined margin were all present.

A matter of especial interest to us all is the method employed in promoting healing when a large surface is denuded of skin. It is obvious that this method is applicable to many cases where the skin is extensively removed by operation or accident. The flaps should be as abundant as possible, their bases broad, and they should be secured upon the raw surface by one or two cat-gut sutures and firm pressure of the superimposed dressings (Fig. 3). There is an insuperable

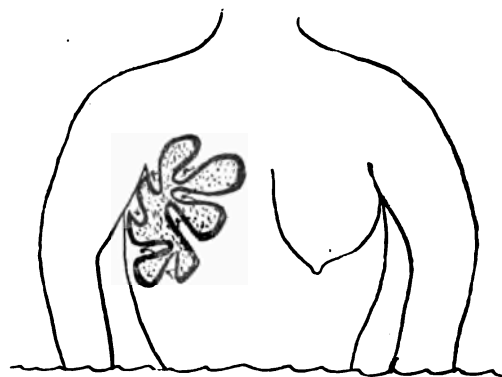


Fig. 3.—Large raw surface covered with multiple skin flaps.

objection to Thiersch's method of implanting large portions of skin. However carefully this is done, some fresh sores are created, which add additional pain and discomfort to a patient already harassed and in suffering from a large open wound. This objection is a very real one to patients who are sensitive and nervous, and is worthy of reflection.

Regarding six other cases of carcinoma which have come under treatment, they presented nothing peculiar. The method of operation was in all cases the same,—removal of the whole breast with the skin and fascia very freely, and also the sternal part of the pectoralis major. This part of the muscle is readily removed, and its ablation gives the most free access to the axilla. On several occasions glands have been detected as large as peas beneath the pectoralis minor, which would have escaped detection by any minor measures.

The following case is also an example of a rare malady—hypertrophy of the mammary glands. Only three examples of this disease were admitted into St. George's Hospital between 1865 and 1895. In none of these was operation needful, so that they were not extreme instances of a complaint which may become dangerous and formidable.

A young woman *æt.* 21, who had been married for one year, and had never been pregnant, applied at St. George's Hospital in May, 1897, on account of pain and increase in size of the *mammæ*. The

did not diminish. Menstruation was normal, and did not seem to influence the condition one way or another. The breasts were well "slung up," and compressed with large pads of wool. Small quantities of fluid were allowed, and dry dietary. Iodide of potassium was administered in full doses. In a month the left *mammæ* had reduced in size, and the right was not so painful and congested. A trial of elastic pressure was now advised, but the patient left the hospital. I have seen the case recently. The breasts are slowly



Fig. 4.—Hypertrophy of the mamma.

enlargement had only been noticed for eight weeks ; they had increased in size very rapidly. The patient was a pale, tuberculous-looking woman. The *mammæ* were of large size, and reached to a point below a line drawn through the umbilicus (Fig. 4). They were marked by large venules, and felt knotty and irregular, as though masses of solid tissue existed. After a fortnight's rest in bed, with purgatives and the free administration of iodide of potassium, the congestion and hardness disappeared. The bulk of the *mammæ*, however,

diminishing under full doses of the iodides. The accompanying photograph, kindly taken by Mr. Clinton Dent, shows well the condition in this case.

Cases of hypertrophy of the *mammæ* differ very much in extent. A number of them seem to be mere exaggerations of the fulness and congestion of the breasts which accompany menstrual engorgements or pregnancy.

The treatment of the minor cases resolves itself into rest in the recumbent position, dry diet,

elevation and compression of the mammæ, and the free administration of iodides internally. Cases are recorded where benefit has occurred in healthy great enlargements of the mamma, associated with checking of the menses from free leeching of the inside of the thighs, or venesection of the saphena vein. Again, the establishment of the secretion of milk by putting an infant to the breast, has in at least one case checked the tendency to enlarge. In some of these obstinate cases, however, no measures seem to check the serious progressive growth of the mammæ, which ultimately may reach to the knees of the patient, and become so cumbrous and hideous as to prevent the patient from moving about. Finally, the skin becomes thickened, warty and rugose, sloughs form from congestion, and profuse discharge may so weaken the patient that she may actually die from the disease, or from the surgical measures taken to effect its removal.

Accordingly, in these exceptional cases operation should never be delayed until the patient has passed into a state of hopeless anæmia and weakness. Instances of true progressive hypertrophy are almost always found in young girls about the time of puberty, or in women under thirty years of age. The diagnosis in bilateral cases presents no great difficulty. In unilateral cases there is a risk of confounding a large firm mammary tumour which displaces the breast substance for hypertrophy. The mass having been removed, a large fibro-fatty or adenomatous tumour is revealed, which might have been taken away, and the mamma itself saved.

In grave cases of the disease, amputation is the sole resource. As a preliminary the masses should be well raised, and subjected to elastic pressure. Skin flaps being turned down, the base is transfixed with long needles, and an elastic cord twined beneath. The tumour is now removed, and any obvious vessels ligatured. When the elastic band is taken off, any remaining points are secured with pressure forceps, of which a large supply should be at hand. Thus very little blood need be lost. As regards the removal of one or both glands at the same time, all must depend upon the circumstances of the case. In great anæmia and exhaustion, an interval should intervene between the two operations.

Removal of the ovaries has been suggested in these cases, but I am not sure it has ever been put

into effect. The result must be somewhat experimental, and if the breasts did not decrease, a serious damage would be done to a young woman.

In very exceptional cases mammary hypertrophy has been present in the male, especially in association with atrophy of the testes, and the large size of the mammæ in some male Zulus and Bushmen is well known.

The microscopical characters of the mammary hypertrophy are very peculiar. Though much consists of masses of fibrous tissue and fat, and little of the gland tissue, masses have been found of a pinkish colour and firm consistence, which are composed mainly of awn-shaped cells, and which suggest sarcomatous tissue. Elaborate microscopical examination of one of these cases in accordance with modern pathological ideas is much to be desired, as observers have hitherto reported many and divers appearances.

True elephantiasis of the breasts, as pointed out by Manson and Dr. Davies of Samoa, is a distinct disease, not to be confounded with true hypertrophy. It has little interest for practitioners in this country.

CLINICAL LECTURES ON URINE.

Delivered at University College Hospital by

J. ROSE BRADFORD, M.D., F.R.S., F.R.C.P.,
Physician to University College Hospital.

V.

GENTLEMEN,—We will consider to-day the nitrogenous constituents of the urine. The only nitrogenous constituents of the urine which are of clinical importance are urea, uric acid, and creatinin; the other nitrogenous constituents of the urine, although numerous, are of no great clinical importance. Creatinin, although present in much greater abundance than uric acid—there is nearly twice as much creatinin as uric acid—and therefore coming next to urea in amount, is of but little importance on account of its solubility. Uric acid derives its importance mainly on account of its insolubility. Creatinin reduces sulphate of copper when boiled in the presence of caustic potash, and so produces a spurious copper reaction. You do not get the bright red deposit that is seen

with dextrose, but it sufficiently resembles this for it to be occasionally mistaken for sugar. Creatinin is present in considerable quantities in all wasting diseases; normally, there is about a gramme present in the twenty-four hours' urine, there being only about half a gramme of uric acid. A gramme, rarely as much as two grammes, is approximately the daily excretion of creatinin. It is mainly derived from the creatin of the food; it is very doubtful whether any is derived from the creatin in the body, but it is undoubtedly derived from the creatin of the food. In wasting diseases the increased excretion of creatinin is probably derived from the creatin of the muscles, and in diseases such as cancer you may have a large quantity of creatinin in the urine. So that the main importance of creatinin lies in the fact that it is a substance producing a spurious copper reaction.

Uric acid is excreted to the extent of about half a gramme in the twenty-four hours in health; in disease there are larger quantities than that, sometimes as much as three or four grammes in the twenty-four hours. Uric acid in healthy conditions is not excreted in the urine as uric acid, it is excreted as a urate, and it exists in the blood also as a urate. You will remember that uric acid is a bibasic acid, and to save one the trouble of repeating its long formula we can represent it in a loose sort of way as H_2U , instead of the long formula which it has. Each molecule of uric acid has two atoms of hydrogen that are replaceable, and the urates represented by the formula M_2U , where M is a monobasic element, do not exist physiologically or pathologically, you can only make that urate of soda by laboratory methods. The biurate, which can be represented by the formula MHU , exists largely pathologically, and another name for it is the acid urate, and a familiar example of this salt is the acid urate of soda, which forms the long needle-shaped crystals seen in gouty concretions. Uric acid is like oxalic acid in the fact that it has these two series of salts, and in addition there are also the quadriurates, which are represented by the formula MHU_2H_2U . This is not simply a mixture of the first two series, it is a definite compound, which is called the quadriurate because there are four replaceable atoms, three of hydrogen and one of the base.

We need not consider here the alkaline urates of the formula M_2U , as they do not exist in physio-

logical or pathological conditions, and the compounds to be considered here are uric acid, the acid urates, and the quadriurates. One of the most important properties of these bodies is their solubility: uric acid is soluble in about one in 14,000 of cold water; the acid urates are twice as soluble as that, and the quadriurates are freely soluble. Uric acid is thus almost insoluble, the acid urates are fairly soluble, and the quadriurates are very soluble. Under physiological conditions uric acid exists in the form of a quadriurate; normally uric acid is present in the blood and in the urine, always in the form of a quadriurate, and the pathology of gout on the one hand and gravel on the other is largely connected with the question of the conversion of the soluble quadriurates into the relatively insoluble biurates or uric acid. A familiar example of quadriurates is the uratic deposit that is found in febrile urine; that is practically pure quadriurates with various bases, but more particularly sodium. If you want to get quadriurates in large quantities you must take the chalky urine of serpents, which also consists of quadriurates. It is an extremely unstable substance; it is decomposed more particularly by water into uric acid and an acid urate, that is an important fact about it. It is decomposed by water in a very simple way, easily understood if you represent it by the formula MHU_2H_2U . You cannot extract quadriurates from the urine by means of water; if you want to observe the properties of quadriurates you must collect the deposit of urates from a febrile urine, place some of the deposit on a filter-paper, wash it with alcohol to get rid of the urine, and in this way you can get more or less pure quadriurates. Then if some of the deposit is placed on a cover-slip, and some distilled water added, you can see the crystals of acid urate forming. One of the most important reactions of the quadriurate is this fact that water decomposes it into the acid urate and uric acid. When you consider the relative solubility, the action of the water is to convert a soluble into an extremely insoluble substance, that is what happens if you decompose quadriurates by the action of water. You can imitate the composition of the serum as regards salts by making up a solution containing $\frac{1}{2}$ per cent. chloride of sodium, and $\frac{1}{2}$ per cent. of sodium carbonate; this mixture dissolves uric acid freely, comparatively speaking;

.5 per cent. of sodium chloride and .2 per cent. bicarbonate of soda will dissolve uric acid freely in the form of a quadriurate. The interesting point is that supposing you make a solution like that, and keep it for twenty-four hours, it decomposes into the biurate, so that if you have a quadriurate in the presence of a liquid containing bicarbonate of soda, when it decomposes it does not split up into a molecule of the biurate and a molecule of the acid urate, but it splits up into two molecules of the biurate. Water splits up quadriurates into uric acid and an acid urate, and this chemical action lies at the root of the pathology of gravel. On the other hand, the splitting up of a quadriurate into two molecules of biurate of soda is at the root of the pathology of gout. In gout the deposits are not of uric acid, but they are the deposits of biurate of soda; in gravel the deposit is uric acid: the two compounds are formed differently from the quadriurates.

The fact of the urine depositing a quantity of uric acid does not necessarily mean that the uric acid is in excess. We see that the amount of uric acid is about half a gramme. You pass sixty times as much urea. The source of the uric acid is more or less unknown; there are a large number of theories, but practically the source of uric acid in the healthy urine is by no means certain. The modern view is that it is derived from the disintegration of proteids containing nuclein, from nucleo-proteids, and that view is based on a large number of physiological facts, and that at the present time is the theory which holds the field. Previously it was supposed that the uric acid excreted was dependent on deficient oxidation,—in other words, that we did not take in enough oxygen to oxidise all the proteid into urea. It has also been supposed to be associated with a particular kind of diet, but in health it is not due to any particular diet. Certain of the carnivora pass little or no uric acid; on the other hand, insects which are not carnivorous pass large quantities of uric acid. You cannot say that in health uric acid depends on diet; it is another matter in disease. You cannot order a diet which will in health lead to no uric acid at all being excreted; it is apparently due to a peculiar metabolism of the nucleo-proteids present in the food and in the body. Even on a diet of pure egg-men there is a certain amount of uric acid

excreted. Therefore it cannot be looked upon simply as a matter of diet; diet of course has an influence upon it, and it has been shown that about three hours after a meal the amount of uric acid excreted increases enormously. At the first sight that might seem to imply that the excretion of uric acid was due to diet; but if you take a meat diet or a mixed diet, or one of white of egg, you still have this great increase in the uric acid excreted about the third or the fourth hour after the meal, and therefore it has been supposed that the uric acid is derived from the immense number of leucocytes which are used up in the process of absorption. There is an enormous increase in the activity of leucocytes during digestion, and to this cause the excess of uric acid excreted has been put down. The important point is that uric acid is excreted in greatest percentage amount some three hours after a meal, and in a healthy condition it does not make any very great difference in the amount whether you have a simple diet, a mixed diet, or a diet of white of egg. Although this is the time when the largest quantity of uric acid is passed, this does not mean that this is the time when there is the greatest risk of uric acid being deposited in the urine. The deposition of uric acid in the urine is most apt to occur in the morning in the starving state before breakfast. I am talking of health, and not of disease; that is the time in the twenty-four hours that the urine is most likely to deposit uric acid; it does not contain more uric acid then, it contains less percentage than at any other time, but the urine passed before breakfast is usually an acid urine. After meals, especially if a large quantity of vegetable matter has been taken, an increased amount of alkaline salts is excreted; and hence, though the amount of uric acid is increased, the salts are increased also, and the uric acid is thus kept in solution as a quadriurate. A starving urine is poor in salts, although the amount of uric acid is comparatively speaking low; that is the period in which the spontaneous deposition in the urine is most likely to occur, and that will illustrate very well that the deposition of the uric acid is not a thing dependent entirely on the quantity present. Febrile urines contain a very large quantity of urates, but it is quite a rare thing to see uric acid in such urines.

Now, in disease you must please keep these two

problems separate : firstly, the amount of uric acid in urine ; and secondly, the precipitation of uric acid as such. Urates are increased in all febrile conditions, for every one knows that one of the first and most obvious effects of fever is to cause copious deposits of these urates. The next most important point is that the amount of uric acid in the urine is very greatly increased in certain diseases of the blood, more particularly in pernicious anæmia, and in the disease known as leucocythæmia. You may have in these diseases as much as six grammes excreted in the twenty-four hours ; these patients take very little food, and you can understand that this great excretion is somewhat remarkable, and to a certain extent it is a confirmation of the view that the uric acid is derived from nucleo-albumins, because the red blood-corpuscles consist largely of nucleo-albumins. It has been taught that diseases of the spleen have an influence on the amount of uric acid. In gout the excretion of uric acid is increased after the paroxysm, and during the paroxysm there is an increased amount of uric acid in the blood, but nobody has ever yet conclusively shown that there is an increased production of uric acid. It has been shown that there is an increase in the blood, and after the paroxysm there is an increased amount in the urine, but it has not been shown that there is an increased production ; it is possible that there is a want of elimination, hence the paroxysm. There is a negative point which is of some interest : in diabetes mellitus the nitrogenous metabolism is increased from the patient eating large quantities of meat, and the uric acid in the case of diabetics is not usually increased ; in gouty diabetes it is increased, but in a great number of cases of diabetes the uric acid is not increased. Garrod showed that the blood of the gouty contains an excess of uric acid, as he obtained crystals of uric acid by soaking a thread in serum acidified with acetic acid. That is a thoroughly established fact. Sir W. Roberts's observations show that when uric acid increases in the blood the alkalinity of this fluid accounts for the formation and precipitation of the acid urate of soda, and what happens in the gouty patient is that the uric acid slowly accumulates in the blood, then it suddenly becomes precipitated in the form of these needle-shaped crystals, and wherever these may be formed they may set up a great deal of irritation. Certain fluids of the

body, more particularly synovial fluid, but other fluids also, cause the formation of acid urate of soda more rapidly than blood does.

To return to the urine, the main importance of uric acid in the urine is the fact that its relative insolubility leads to its being precipitated as uric acid ; all urines, if they are prevented from decomposition, precipitate uric acid sooner or later, in health or in disease ; but in health the precipitation of uric acid—that is to say, the conversion of the quadriurate into uric acid—does not occur within any short time of the urine leaving the body, sometimes it is twenty-four hours or longer. If the ordinary urine is left in the air, ammonium carbonate is formed, and then you have urate of ammonia, and the precipitation of uric acid does not occur. In certain diseased conditions the urine deposits uric acid very much sooner, and, in fact, sometimes the urine deposits uric acid before it has left the pelvis of the kidney, so that you have a stone in the pelvis of the kidney, or you may have a stone in the kidney, or the precipitation may occur a few minutes after passing the urine. In health, as mentioned above, this condition does not occur till a considerable time has elapsed after passing the urine. In disease the conversion of quadriurate into free uric acid occurs sometimes before the urine has left the kidney or the bladder, and the patient then runs a considerable risk of forming a urinary calculus or renal calculus ; and I may as well say that most cases of stone in the bladder arise secondarily to stone in the kidney, first of all there is a small stone in the kidney, and that passes into the bladder and forms the nucleus for a second or larger stone. What are the conditions which will lead to the formation and precipitation of uric acid ? First of all you have to consider the amount of uric acid. It is a platitude to say the more uric acid there is in the urine the more likely is this change to occur, that is self-evident, and to people who have this abnormal tendency a large meat diet is injurious ; but there is a point about a large meat diet which is sometimes forgotten. A large meat diet may lead to precipitation of uric acid simply on account of the presence of phosphate of soda in the meat ; the acidity of meat is also partly due to lactic acid, but a meat diet renders the urine acid owing to an increased excretion of acid phosphate of soda. Thus an excessive meat diet may

affect the uric acid in the urine by increasing the amount excreted, or indirectly by affecting the acidity of the urine by increasing the acid phosphate of soda. If the urine is acid, this breaking up of the quadriurate occurs with much greater rapidity; in the normal urine it does not occur rapidly, because the normal urine contains a lot of salts; but if it contains acid salts, the urine acts practically like water, and splits up the quadriurate, and that is the real reason why the starving urine before breakfast is most apt to deposit uric acid, it is the urine of the metabolism of the tissues unaffected by diet. The urine before breakfast is passed at a long interval after meals, and is very acid, and therefore it is the urine most prone to deposit uric acid.

The proper way to treat a patient with gravel is to give a large dose of citrate of potash when the patient goes to bed, so as to render the morning urine as alkaline as possible, and order him a suitable diet during the day to render the diurnal urine less acid or alkaline. The next thing that helps the precipitation of uric acid, apart from the amount of acid present, is that the more dilute the urine or the more scanty the presence of salts, the greater the tendency to precipitation, and it is well known that the urine of granular kidney is a urine that tends to deposit uric acid. That little point will show you that the question of the dilution of the urine is at any rate of equal importance with the quantity of uric acid present. In pneumonia, where a concentrated urine is passed, the amount of uric acid is considerable, it all passes out as urates, and is therefore soluble. Take, on the other hand, a patient with granular kidney, whose urine is very dilute, with very little uric acid, and yet that patient may deposit enough uric acid to give him serious trouble in the way of gravel or even of stone. There is another thing that influences the precipitation of uric acid, and that is the amount of pigment present in the urine. These are the main points of interest; and when you think that gout and gravel practically depend on the conversion of quadriurates on the one hand into acid urate of soda, and on the other hand into free uric acid, I think you will admit that it is of importance. There are few diseases which can be explained satisfactorily; scabies is one, and gravel and the formation of tophi in gout are others. No one can say with certainty why a

person with gout has an excess of uric acid in the blood, but given that condition one can explain the rest of the phenomena. There is an interesting corollary; no one has ever heard of a patient suffering from an attack of gout and gravel at the same time, although of course a person with gout is liable to gravel; but you never have a person with a paroxysm of acute gout passing gravel at the same time, and when you see that gout is due to the presence of an excess of uric acid in the blood, and gravel to the precipitation of uric acid in the urine, you can understand why you do not have the two things occurring together.

Do not forget that uric acids, and urates, and creatinin have the power of reducing copper; it is not limited to creatinin. As regards the test for uric acid, clinically one relies practically on the crystals; one is not in the habit of doing the chemical tests, and there is not much to be said about the crystals; if you want to get the crystals, you add some hydrochloric acid and keep the urine for twenty-four hours, under these circumstances it crystallises out, and one of the characteristic facts is that the crystals are coloured, and they are coloured by the uroerythrin; pure uric acid is of course colourless, and it crystallises in flat colourless plates. If you want to prepare the pure specimen you must prepare the impure form first, and then dissolve it in some alkali, and then re-precipitate it again. If you take crude uric acid and dissolve it up in an alkali, and then recrystallise it, it does not crystallise in the well-known spindle-shaped coloured form that every one is familiar with, but it crystallises in these flat colourless plates.

Occasionally acid urate of soda and acid urate of ammonia are formed in children; these are not present in normal urine, but in children's urine these two salts are sometimes formed, and these bodies crystallise, forming so-called hedgehog crystals,—a round central body with spikes sticking out. The delicate urethra of children is liable to be injured by these prickles, and they will sometimes cause retention of urine; they irritate the urethra and cause a spasm of the urethra, and it is a condition easily put right by giving an alkali: it is not of any importance in adults.

As regards *urea*, urea is of comparatively little importance; I suppose it is somewhat of a heresy to say this, but though urea is of course of great scientific importance you do not arrive at very

great diagnostic results by studying the excretion of urea in the urine. Urea is affected enormously by diet; the thirty grammes that we usually pass, although this amount is liable to considerable variations, is mainly derived from the food. If you examine the urine of a starving patient—ovariotomy patients are usually starved for a day or two, and they are very frequently comparatively healthy people except for the presence of the tumour—you will find that they do not pass more than ten or twelve grammes of urea in the twenty-four hours. It is very unfair to take the thirty grammes as the amount of urea that a hospital patient ought to pass; thirty grammes is what a healthy person should pass. If you lie in bed all day on a scanty diet you are pretty sure not to pass thirty grammes of urea; therefore, if a patient does not pass thirty grammes in the twenty-four hours you must not assume that there is anything very seriously wrong with him; thirty to forty grammes is the amount for a healthy person, and is not the amount for a patient lying in bed on a sick diet. On the other hand, the appetite fails in most diseases, and hence a patient who is passing as much as thirty grammes may be losing much of his nitrogenous tissues; it is quite possible he is eating very little, therefore the estimation of urea is useless unless you know what the nitrogenous intake is.

CLINICAL OBSERVATIONS ON SCURVY IN EARLY LIFE.

BY

G. A. SUTHERLAND, M.D.Ed., M.R.C.P.Lond.,
Physician to Paddington Green Children's Hospital.

PART I.

It is now almost twenty years since Dr. Cheadle drew attention to this subject in a paper entitled "Three Cases of Scurvy supervening on Rickets in Young Children." His clinical observations were subsequently fully confirmed by the pathological investigations of Dr. Barlow, and the views expressed by these two writers have been very generally accepted in this country. In 1892 the first mention of the subject in America came from Dr. W. P. Northrup, who expressed the opinion that the affection was extremely rare in that country.

His paper, however, was evidently the means of directing attention to the disease, with the result that in the beginning of 1895, 106 cases of scurvy in children were reported at a meeting of the Academy of Medicine in New York. Valuable papers have been published on the subject by Northrup and Crandall, Fruitnight, Ling Taylor, and others, which show that American experience and American opinion support the views first enunciated in England. In Germany a good deal of attention has been paid to the subject of scurvy in children. The theory of "acute rickets" has been dropped, and the scorbutic nature of the affection has been recognised by Baginsky, Rehn, Heubner, and others, who have recorded cases very similar to those met with in England. Other German writers (Fürst, Starck, Conitzer, Hirschsprung, &c.) do not accept the English views on scurvy, but hold that the special symptoms associated with rickets are dependent on a hæmorrhagic diathesis, the nature of which has not yet been thoroughly ascertained. Dr. Fürst mentions that no case of infantile scurvy has been published in Russia, although cases of scurvy amongst adults are common in that country. In Sweden a case of infantile scurvy was recorded by Dr. Ingerslev as early as 1873, and it is perhaps to him that the priority in the recognition of the disease is to be given. In Holland fifty cases of "Barlow's disease" have been published (Fürst), but I have not been able to consult the original papers. Cases have also been reported from Canada, Australia, and India.

A variety of names has been applied to scurvy in early life; in the first place, by the original writers to whom the nature of the affection was not clear, and more recently by others who deny that the disease is really scurvy. The earliest name, "acute rickets," has now dropped out of use even in Germany. "Scurvy rickets" refers more particularly to the common association of these affections; but as they are really two distinct diseases, the name is deservedly falling into disuse. "Infantile scurvy," a title used by Ingerslev, Jalland, and Barlow, is to a certain extent misleading, as it suggests a different affection from juvenile, adult, or senile scurvy. As Dr. Barlow has laid stress on the fact that the disease is one and the same at whatever period of life it occurs, and that age is only a modifying factor as regards certain sym-

ptoms, it seems to be less ambiguous to retain the term "scurvy" alone. "Osteal or periosteal cachexia" (Gee) and "hæmorrhagic periostitis" (Thomas Smith) were provisional titles employed before the disease was fully understood. "Osteopathia hæmorrhagica" has been suggested by Conitzer, who does not recognise the disease as scurvy. In Germany, and also by some writers in this country, a title has been adopted of an indefinite character, such as "rickets with a hæmorrhagic diathesis," or "hæmorrhagic rickets" (Fürst), or "a scurvy-like disease of rachitic infants" (Heubner). All of these ignore the fact that scurvy occurs quite apart from any rachitic manifestations. Finally, there are some complimentary titles of German coinage, namely "Barlow's disease," "Möller's disease," "Cheadle-Barlow's disease," "Möller-Barlow's disease," which do not bear translation well, and only serve to confuse the student in consulting the literature of the subject.

Ætiology.—In regard to the scurvy of adults, the question of ætiology was settled by the results of treatment, and the same method has to be adopted in the case of children. It is essentially an affection associated with a diet defective in sound fresh animal or vegetable products. What the exact defect is, whether potash, or organic acids, or a combination of these, has not yet been definitely determined. It is, however, sufficiently clear that the increase of scurvy amongst children during the last twenty years has been largely due to the substitution of various artificially prepared infants' foods, for breast or cow's milk and natural vegetable products. In the case of scurvy in infants this cause is almost invariably present. In the case of older children it is much less important, and another factor frequently comes in—namely, a positive dislike to fruits and vegetables, which leads to the omission of those substances from the diet. As the diet is of the greatest importance in the ætiology of scurvy, it is necessary to consider, firstly, what foods are safe, *i. e.* contain antiscorbutic properties; and, secondly, what foods are unsafe, *i. e.* do not contain antiscorbutic properties.

Breast milk from a healthy mother is usually considered the best food for the first nine months of an infant's life, and so far no case of scurvy has been recorded which would tend to alter this belief. If lactation is too prolonged it is possible

that the changes in the milk as time goes on may so alter its quality that scurvy may develop. Two cases of this nature have been reported from America, in both of which the infants were suckled for fifteen months, when symptoms of scurvy appeared. A change of diet to fresh cow's milk and vegetables led to rapid recovery.

Fresh unaltered cow's milk is also regarded as a complete diet for infants, and clinical experience in connection with scurvy supports this view. In the absence of any clear evidence of the development of scurvy on a full milk diet, and in the presence of the therapeutic evidence which proves that many cases of scurvy in infants have been cured solely by the administration of fresh cow's milk, we are justified in regarding cow's milk as distinctly antiscorbutic. At the same time it is not to be regarded as possessing antiscorbutic properties to the same extent as fruits and vegetables do. Hence the quantity as well as the quality of the milk is important, and over-dilution must be avoided, as the total amount of antiscorbutic material administered *per diem* might not come up to an infant's requirements. The practice of boiling or scalding milk on delivery in order to prevent fermentation is now very generally carried out, and there is no evidence to show that scurvy has ever developed on a diet composed of milk thus treated, when the process has been the ordinary one of simply bringing the milk to the boil, or mixing it with boiling water. The boiling of milk may act to a certain extent in lowering its antiscorbutic value, but the effect is not of such importance as to be considered an ætiological factor in scurvy. Similarly the process of Pasteurisation, in which the milk is subjected to a temperature of 167° F. for twenty or thirty minutes, does not appear to materially alter its antiscorbutic properties.

Within the last few years other modifications of milk have come into use, and the process of sterilisation has been carried out on a large scale in Germany, America, and more recently in this country. By sterilisation is meant the exposure of the fresh milk to a temperature of 212° F. or upwards for a period of twenty minutes or longer. Several examples of scurvy have occurred under the use of this food. One of the very worst cases I have met with was in an infant who was fed exclusively for the first three months on sterilised milk, and then for three weeks on a patent food

and water. At the end of that time scurvy developed; and although the patent food was undoubtedly an important factor in inducing the scurvy, still the period in which it was employed was so short that I believe the illness was primarily dependent on the exclusive use of sterilised milk. Drs. Northrup and Crandall, Herbert Menzies, and Louis Starr, have published similar cases. Dr. Starr's series of five cases is of great interest, because in the treatment he continued the same food, but stopped the sterilisation, and added a small quantity of raw beef juice and orange juice, with the result that all the patients made a rapid recovery. It would appear that prolonged sterilisation at very high temperatures considerably impairs the antiscorbutic properties of milk, and may reduce them so low as to render the milk an unsafe food. Similar results have followed from the prolonged use of peptonised or pancreatised milk, a diet which, quite apart from the risk of scurvy, is not one to be recommended.

The diet to which the vast majority of the cases of scurvy in early life can be traced, is one consisting entirely, or almost entirely, of one or more of the preserved and artificially prepared infants' foods. These foods may be divided into three classes: (1) preserved milk—condensed, powdered, or powdered and malted; (2) starchy food, unchanged save by the process of cooking, or partly converted into glucose or maltose or soluble starch; and (3) a mixture of milk and starchy substances. In some of these foods, *e. g.* the milk preparations, the antiscorbutic element was originally present; in others, *e. g.* the dried cereals, it was absent or reduced to a minimum. In all, during the process of preparation the element of freshness was destroyed, and along with that the antiscorbutic property vanished.

Opinion is still divided as to the value of meat, raw meat juice, or beef tea in preventing the onset of scurvy. It must be remembered that meat is eaten in this country after it has been hung for some time, and that consequently it is not, strictly speaking, fresh. Dr. Ralfe has pointed out that the reaction of freshly-killed meat is alkaline, and after *rigor mortis* has passed off it becomes acid, owing to the formation of lactic acid. Clinical evidence shows that scurvy may develop when meat has formed a part of the regular diet, and that scurvy may persist when meat has been added to the

previous diet; so that it seems probable that meat cannot be regarded as a safe food from the antiscorbutic stand-point.

Reference has now been made to the various foods most commonly employed in the feeding of infants, and their action as scurvy producers. Objection has been taken to the view that certain of them directly induce scurvy, on the ground that many thousands of children have been brought up on similar diets and have never shown any signs of scurvy. In reply to this, Dr. Cheadle has referred to the fact that amongst the poor potatoes are usually given to children at a very early age, it may be as a regular part of the diet, or as a small share from the family table; and as potatoes are powerfully antiscorbutic, the food elements which are necessary to avert scurvy are thus supplied. Similarly, a little fruit or vegetable soup is occasionally administered, which although small in quantity is sufficient to ward off scurvy. It cannot, however, be asserted in the present state of our knowledge that every infant fed solely on preserved food, even if it is apparently entirely devoid of antiscorbutic elements, will develop scurvy after a certain length of time. There may be some hereditary diathesis about which at present we know nothing, and there certainly are predisposing circumstances, which will be referred to later. That there are some unknown factors in the ætiology of scurvy seems probable from a case recorded by Dr. Wallis Ord. An infant aged eleven months developed scurvy, which eventually proved fatal; while a twin brother, brought up on exactly the same diet, presented no signs of the disease.

Summing up these observations on the ætiology, we find—

1. That scurvy in children is a disease of defect, not of excess.
2. That the defect consists in the absence from the diet for a prolonged period of a sufficient amount of the antiscorbutic element in food.
3. That although we do not know exactly what this element is, we know the foods which contain it.
4. That the antiscorbutic element is found most abundantly in fresh or living food, more especially vegetables and fruits, and that the further we get from fresh food as the regular diet, the greater the tendency to scurvy.
5. That there is a class of prepared foods for infants in which, during the process of prepara-

tion, the antiscorbutic element has been entirely destroyed.

Predisposing Circumstances.

1. Preceding illnesses. A history of prolonged diarrhoea, or of diarrhoea alternating with constipation, or of persistent sickness, is present in many cases of scurvy. Sometimes the onset of scorbutic symptoms follows immediately on an attack of acute intestinal disturbance, or a specific fever, or the process of dentition in a weakly infant.

2. The season of the year. Scurvy is more prevalent in the winter than in the summer months, and the period of early spring supplies the greater number of cases. Out of a total of sixty-five cases I have collected, in which the month of onset is mentioned, eight occurred in January, five in February, six in March, fourteen in April, seven in May, two in June, three in July, two in August, three in September, eight in October, five in November, and two in December.

3. Age. The overwhelming proportion of cases occurring during the first two years of life is very striking. In a series of 135 collected cases, I find that five occurred during the first six months, fifty-two from the sixth to the twelfth month, fifty-five from the first to the second year, and twenty-three between the ages of two and twelve years.

(To be continued.)

POST-OPERATIVE INSANITY, ESPECIALLY IN WOMEN.

By W. P. MANTON, M.D.

ALTHOUGH it had long been known that mental derangement sometimes follows in the wake of surgical operations, no especial attention had been directed to this subject, particularly as regards women, until the publication of Thomas's paper in 1889. In this article twenty-six instances of acute mania, melancholia, and hypochondriasis, the sequelæ of gynæcological operations, were reported—six of the cases having fallen under Thomas's personal observation, the remaining twenty being collected from various sources.

At that time post-operative mental alienation was believed to be of rare occurrence, but the lected experience of many observers during the

past eight years has shown that, while not frequently met with, the condition is by no means uncommon, while the increasing literature of the subject—the latest article in this line contains a bibliography of no less than sixty-six distinct titles, and this does not by any means exhaust the list—has greatly augmented our knowledge of the ætiology and course of the mental disorders resulting from surgical procedures. The discussion of this condition, of course, properly falls within the domain of the mental specialist; but as the beginning of the attack occurs usually while the patient is still under the care of the surgeon or gynæcologist, it will not be entirely foreign to our department in medicine if we devote a little time to its consideration.

I shall confine my remarks to a brief summary of the following points:

1. The class of cases in which post-operative insanity is most likely to develop.

2. The differentiation of this psychic disorder from insanity arising from other conditions.

3. Its frequency.

1. While it is claimed by some that psychic disturbance is as likely to develop in mentally sound individuals as in those who possess a latent defect, a study of the subject leads me to believe that in nearly every instance careful investigation will discover some acquired tendency or hereditary taint which predisposes to the mental unbalancing. In such instances, it can be readily understood, the mental impression of an operation, which, as pointed out by Dent, may arise by anticipation, by the actual operation, or by its after effects, engrafted upon an unstable mental organisation may so disturb the equilibrium of the mind as to give rise to confusion and chaos. The actual shock of the operation added to the previous mental strain of anticipation, the lowering of the vital energies through loss of blood, or the entrance into the economy of specific poisons—septic material, carbolic acid, iodoform, cocaine, and the like—must surely act more profoundly on the central and peripheral nervous systems of the tainted individual than on one who is physiologically sound. It would also seem most reasonable to suppose that the more delicate and susceptible nervous organisation of woman and her greater tendency to neurotic disorders would render her more liable to mental derangement following

operations than the opposite sex, and we are, therefore, not surprised to find that the greater number of cases of this kind reported have occurred among females. Simpson states that "if gynæcological work be excluded, the preponderance of the female sex as sufferers in this direction disappears." I have no statistics of general surgery at hand, but from my experience in hospital and general work I should say that general surgery on the female is very much less frequently carried out than on the male, so that I cannot see why, in attempting to determine the ætiology and frequency of post-operative insanity, gynæcological operations should be excluded.

It must not be overlooked or forgotten in the discussion of this subject that in some cases of insanity following operations the surgery has nothing whatever to do in the production of the mental symptoms. For, as Clevenger remarks, traumatism may precede insanity and still have no relation to the insanity. In not a few cases recorded as post-operative, the mental symptoms have not manifested themselves for so long a period subsequent to the surgical act that it is only by a stretch of the imagination that the two can be connected as cause and effect.

It may, therefore, be put down as a rule that post-operative insanity is most likely to develop in those subjects who have a bad personal history or are handicapped by heredity, and the operator should proceed with the utmost caution in his treatment of this class of cases.

2. As regards the second point under discussion, it may be stated at the outset that without a previous knowledge of the determining moment no one would be able to distinguish, from the symptomatology, post-operative insanity from the insanities arising from other causes.

Vene gives the average time of the onset of the attack as the second to the fifth day, but it may appear suddenly immediately following the operation, or develop slowly, so that several weeks may elapse before the mental symptoms become serious. Sears found that the type assumed by this class of cases was, in 186 instances, of the acute confusional variety, but a perusal of the literature shows that in a large percentage of cases the form is the acute maniacal or melancholic.

Most of these post-operative cases run a rapid course, and the tendency is to recovery; a few die,

and probably fewer still terminate in a chronic condition.

3. From the records of 5500 surgical cases treated in the wards of Annandale, of the Edinburgh Royal Infirmary, Simpson found ten cases of mental derangement which could be ascribed to operative causes. Homans saw two cases following 1000 abdominal sections for various conditions. These figures probably represent fairly accurately the proportion of post-operative among all the insanities. On account of the fact that most of these cases of post-operative insanity either recover or die within a comparatively short period after the onset of the symptoms, few becoming chronic, asylum statistics furnish comparatively little information regarding the condition. I have been at some pains to examine the records of the Eastern Michigan Asylum, and find that in about two thousand patients, whose histories I have looked over, in only two cases, both females, is the cause of the mental trouble ascribed to operative influence.

The histories of these cases are as follows.

1. H—, widow æt. 62, mother of two children. Has an insane second cousin. Patient has always been frail and delicate. Following her last labour suffered from uterine prolapse, for the relief of which she has worn a pessary and abdominal supporter. Five months before admission to the asylum she underwent an operation for mammary cancer of the right side. Very soon afterward mental symptoms developed, which finally led to her transfer to the institution. On admission she was found to be suffering from *agitated melancholia*. The distressing symptoms continued for several years, but towards the end of life were somewhat ameliorated. She died five years from date of admission.

2. F—, married, æt. 32, mother of five children, has an insane brother. During the spring previous to admission patient had a miscarriage, and was sick or ailing all of the following summer. Late in the fall she was operated on for laceration of the cervix and perinæum. Mental symptoms developed two weeks later, and after two months of home treatment she entered the asylum, suffering from *agitated melancholia*. She was then restless, sleepless, moaning, crying, and apprehensive. She feared personal injury or another operation, and fancied that the uterus had been removed and the

bowels sewed together. She was also suicidal, and later became destructive of furniture and clothing. Her physical condition has slowly improved; she sleeps better, and there are days during which she is comparatively comfortable, but she has little appreciation of her condition, and the mental symptoms continue unimproved. —*Annals of Gynecology and Pediatrics*, Sept., 1897.

NOTES.

Aikins' Hoop-Iron Splint in Fractures of the Humerus.—This splint has been used with much satisfaction for many years by the inventor, Dr. W. T. Aikins, of Toronto, and by a large number of his old pupils; but a description of it has never been placed on record. The material used is the ordinary band or hoop-iron, one inch wide for young children, and from one and a quarter to two inches wide for an adult. The material can be shaped by hand or by a pair of pliers or monkey-wrenches. It is at first so bent and twisted that an arch is formed over the top of the shoulder, the anterior limb reaching downwards over the front of the chest in a direction approximately towards the umbilicus, and may be from eight to twelve inches in length. The posterior limb forms the vertical part of the splint, and passes down the back part of the shoulder and arm to a point about an inch below the point of the bent elbow. At this level the iron is again bent somewhat obliquely, so as to pass under the forearm in a direction towards the mid-line of the body. The splint thus almost forms a triangle, the sides of which, however, do not lie in the same plane. The distinctive feature is that the distance from the shoulder arch to the bend at the elbow should be such that when the arch is fixed in position by strapping, considerable extension can be made on the muscles of the arm by drawing the forearm down to the horizontal limb of the splint below the elbow. In applying the splint, after being well padded, it is placed with the arch over the shoulder and secured very firmly (but quite free) by one or two strips of rubber adhesive

strapping about an inch and a half broad, and long enough to reach well down upon the back and front of the thorax. For the sake of additional security, one or both straps may be given a turn round the top of the arch. Similar straps are made to embrace the anterior limb of the arch, and pass horizontally round the thorax. A moulded splint for the upper surface of the forearm should be very carefully padded and fitted, and then bound down to the horizontal limb of the splint by strapping, thus setting up very effectual counter-extension. If desirable short coaptation splints may be applied over the fracture.

The advantages claimed for the splint are—

It is the only splint described which furnishes in itself the means of making effectual extension of the muscles of the arm.

It is cool, light, and affords ready means of examining the parts during healing with the minimum of disturbance.

It permits the direct application of evaporating lotions or the cold coil where indicated.

In compound fractures ready access may be obtained to the wounds, and if the iron of the splint be brushed over with melted paraffin it can be readily rendered aseptic, and lotions and discharges leave it unaffected.—*Montreal Med. Journ.*

Infantile Diarrhoea and its Treatment by Sterilized Water.—Watu advocates the treatment of infantile diarrhoea by a regimen of boiled water, cooled to a suitable temperature, and given in small quantities every hour or half-hour, or as thirst demands, to the exclusion of all food for eight, twelve, or even twenty-four hours. By diluting the irritating secretions, dislodging the debris of decomposed alimentary matter retained in the folds of the mucosa of the canal, increasing the pressure in the blood-vessels, and dissolving the toxins attached to the formal elements, the ingested water carries away and eliminates the poison from the system, changes the morbid character of the stools, and effects a reduction of the temperature and recovery, especially when administered in acute cases at their commencement and in children not very young.—*Charlotte Medical Journal*, August, 1897.

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CLINICAL OBSERVATIONS ON SCURVY IN EARLY LIFE.

BY

G. A. SUTHERLAND, M.D.Ed., M.R.C.P.Lond.,
Physician to Paddington Green Children's Hospital.

PART II.

Clinical Signs and Symptoms.

Scurvy is a disease in which the premonitory symptoms usually pass unrecognised. It is also associated in many cases with rickets, the signs of which are more evident, and consequently the scorbutic manifestations are overlooked until the onset of hæmorrhage, or excessive tenderness in the limbs, excites suspicion of some further trouble. At the outset, a gradually increasing listlessness with debility comes over the child; instead of running about actively he will prefer to sit still or to lie down; or, if an infant, the patient will show none of the excessive activity of the limbs which is characteristic of that period of life. As the disease advances the signs of anæmia increase; the child becomes short of breath on exertion, and the complexion assumes a dirty yellow hue with pallor of the lips and conjunctivæ, and of the skin generally. The listlessness increases to complete apathy, the patient taking no notice of what is going on around, refusing to join in the play of other children, and being fretful and irritable if disturbed. This is the condition known as "scorbutic cachexia," and from it alone the disease can often be easily recognised—the prostration being much greater than in other forms of anæmia. Finally, and it may be suddenly, there comes a total cessation of voluntary movements in the lower limbs—a condition of *pseudo-paralysis*; the child screams if he is moved or touched, and may lie moaning even when undisturbed, and the fear of being touched may be so excessive that he cries out when anyone approaches him. With this limb affection, or in some cases without it,

hæmorrhage may occur about the gums or the eyes, in the skin or in the subcutaneous tissues, or from the bowel or bladder. If the disease is left untreated, the swelling and tenderness of the limbs increase, the child becomes paler and more apathetic, the hæmorrhages continue, and death ensues from exhaustion, or loss of blood, or some complication such as pneumonia or bronchitis. On the other hand, if proper treatment is employed sufficiently early, the recovery is, as a rule, rapid and complete; the swelling of the limbs diminishes, the pain and tenderness pass off, voluntary movements are performed, the spirits revive, and the child soon loses every trace of acute illness.

The changes present in connection with the long bones are of extreme importance, because these are in children the most prominent, the most frequent, and the most characteristic of all the signs. As in adults sponginess of the gums is the most constant sign of scurvy, although by no means invariably present, so in children and more especially in infants, swelling of one or more limbs due to subperiosteal hæmorrhage is the most important diagnostic sign, although it is not invariably present.

The onset of the limb affection is marked by pain and tenderness developing suddenly in the extremity of one or other long bone, most commonly at the lower end of one femur. The child is observed to keep the affected limb at rest, and to cry out when it is touched or moved. Careful examination at this stage will usually show a slight thickening around the end of the bone near the epiphysis. This thickening increases, sometimes slowly, sometimes rapidly, and extends along the limb until the bone in the whole course of the diaphysis may be affected. In other cases the swelling commences at some intermediate part of the diaphysis, and spreads gradually towards the extremities of the bone. From post-mortem evidence we know that this swelling is due to hæmorrhage occurring at the epiphysis or from the under surface of the periosteum.

The limb at the part affected is swollen, tense, and glistening. There is no pitting on pressure

and no local increase of temperature. The swelling may occupy the whole extent of the limb between the two joints, but the joints themselves are neither swollen nor otherwise affected. The pain and tenderness may be so great that it is impossible to make a thorough examination, and in cases in which the diagnosis is doubtful—for instance, if the presence of pus is suspected—it is advisable to put the child under chloroform. The subcutaneous and muscular tissues may be more or less infiltrated with blood, but on deep palpation it is easily made out that the real cause of the swelling of the limb lies in the bony affection. Along the course of the bone and surrounding it is a firm, fixed swelling, which is limited at the extremities by the epiphysal attachments of the periosteum. If the effusion of blood occurs primarily at the epiphysal ends, the form of the swelling will be that of two cones with their apices meeting; while if the effusion is primarily in the centre of the shaft, the form of the swelling will be that of two cones with their bases in apposition. In some cases deeply-seated fluctuation can be made out, but as a rule the tension of the detached periosteum is so marked, and the depth below the skin is so great, that this sign cannot be depended on. In severe cases the epiphysis is separated from the shaft, and crepitus may be elicited between the opposed surfaces. Fractures may occur in the shaft of the bone, either spontaneously or from handling of the limb, and such fractures show little or no tendency to heal as long as the disease continues unchecked. The liability of the diaphysis to fracture is explained by the fact that being separated from the periosteum it is deprived of a strong support and of an important blood supply. If the condition is maintained, the liability to fracture is still further increased by the atrophy of the shaft which ensues.

The pain is caused by the tension of the raised periosteum, and is so great that in the early stages the child avoids all movement of the limb affected; and as the swelling increases, a condition of *pseudo-paralysis* is rapidly developed. The limb then lies motionless in the position of least discomfort, which is that of semiflexion and eversion. Œdema, commencing at the extremity of the affected limb, is often present, and bears a direct relation to the amount of periosteal distension present. In the early stages the œdema is dependent on the interference with the flow of blood caused by the

swelling around the bone, and is often unilateral; but in the later stages it may be traced also to the intense anæmia and cardiac weakness which supervene, and is usually bilateral.

The periosteal hæmorrhage usually affects the corresponding bones on the two sides, but there is no complete identity either as to the extent of the lesion or the date of onset. The lower end of the femur, as already stated, is the part most frequently affected, possibly because there is a larger blood supply and greater activity of growth in this than any other bone. The other long bones which are common situations for subperiosteal hæmorrhage in scurvy are the tibia and fibula (both ends), the humerus (usually the upper end), and the radius and ulna, especially the lower ends. The ribs also may be affected, especially at their anterior extremities. In severe cases it may happen that the ribs become detached at the costo-chondral articulations, with the result that the sternum and its cartilages drop backwards, and during inspiration the depression in the chest-wall thus produced is increased still further. The deformity is very marked, but complete restoration usually takes place if the patient recovers.

As a rule, only a few of the long bones are affected by these subperiosteal hæmorrhages, but in severe cases the condition may become general. One of the worst cases I have met with was that of an infant aged four and a half months who had been acutely ill for three weeks, the illness commencing with swelling and loss of power in the right upper arm. On admission it was found that all the long bones of the upper and lower extremities were more or less affected. The swellings were irregularly distributed over the regions of the long bones, being in some most marked in the course of the shaft, and in others at the epiphysal ends. The right humerus was fractured in two places—in the middle of the shaft and at the lower epiphysis. In addition the following bones were fractured in the course of their shafts, namely, both femora, the left humerus, the left ulna, and the left radius. These fractures were complete, the broken ends being freely movable on each other, and considerable distortion of the limbs had occurred. Under suitable treatment the swellings in the limbs subsided, the effusion was absorbed, and all the bones united firmly. There was no sign of rickets in this patient throughout

the whole course of the illness. Considerable confusion as to these bone lesions has arisen from the use of the term "scurvy-rickets," as implying a single disease, whereas, for clinical purposes, a distinction must be made between (1) scurvy, (2) rickets, and (3) scurvy associated with rickets. As far as my experience goes in scurvy in infants hæmorrhage may occur under any part of the periosteum, and when the shaft is completely separated from the periosteum by blood, complete transverse or oblique fractures may be produced merely by handling the limb—the so-called spontaneous fracture. Such fractures may be at the epiphysis or in the course of the shaft. In rickets the process consists of a softening and bending of the long bones, with possibly greenstick fracture of the shaft. In scurvy associated with rickets, the hæmorrhage is usually near the epiphysis, which becomes separated or easily separable, and even if the effusion extend along the shaft the bone does not appear to have the same brittleness as in uncomplicated scurvy, and fracture of the shaft is not common.

The bones of the trunk as well as of the extremities may be affected in scurvy. In connection with the skull, extravasation of blood may occur below the pericranium, producing a prominent fluctuating swelling, which in the early stages is extremely tender. This may develop spontaneously or as the result of an injury. A very marked series of symptoms is associated with hæmorrhage between the orbital plate of the frontal bone and the subjacent periosteum. The first indication of this occurrence is œdema of the eyelid, which may be present in one or both eyes, and affects chiefly the upper eyelid. In more severe cases the eye will be completely closed owing to the amount of swelling present, a condition usually accompanied by actual hæmorrhage into the tissues of the lid, so that it is at first red, then black, and then yellow, as the effused blood undergoes absorption. Bleeding may also occur under the conjunctiva, which becomes swollen and ecchymotic. Proptosis is present in those cases in which the hæmorrhage under the periosteum of the orbit is sufficiently extensive to displace the eye forwards and downwards. Intracranial hæmorrhage is occasionally met with, usually from the dura mater, and more rarely in the substance of the brain. The onset of the bleeding may be marked by a convulsive

seizure, and if the effusion is not extensive, the symptoms may entirely pass off. In other cases of repeated and extensive bleeding the symptoms are those of gradual cerebral compression with irritation.

In some cases angular curvature of the spine has been noted in association with scurvy. Dr. Ling Taylor has described a well-marked example of scurvy in an infant eleven months old, who presented a bent and rigid spine with a distinct projection at the first and second lumbar vertebræ. The suggestion of Mr. Howard Marsh that such curvatures may be due to hæmorrhagic destruction of the bodies of the vertebræ, is rendered probable by the fact that this condition was found by Dr. Barlow in a case on which he made a post-mortem examination. The presence of spinal changes due to rickets will be a predisposing factor in the production of these spinal hæmorrhages. Bleeding may also occur under the periosteum of the scapulæ and the iliac bones, producing tender swellings as in other parts.

The leading sign of scurvy in adults—namely, swelling and sponginess of the gums, does not occupy the same prominence in connection with scurvy in early life. In infants who have not cut any teeth, the gums are usually anæmic, and minute hæmorrhages may occur over the site of the oncoming teeth; but further than this no local changes are visible. In infants who have cut some teeth, a red swelling appears in their neighbourhood, which may increase to such an extent as to form a large protuberant mass of gum tissue, purplish in colour, and entirely concealing the teeth. The swollen tissue is apt to ulcerate, and bleed on the slightest touch, and the breath may become extremely offensive. The teeth may be detached from their sockets and forced outwards by extravasated blood, so that they lie loose in the spongy tissue or drop off. In rachitic infants in whom the process of dentition is delayed, the affection of the gums is usually trifling or entirely absent. After the age of two years the gum symptoms begin to assume a greater prominence, and the periosteal ones at the same time become less severe and less frequent, as Dr. Barlow has pointed out.

Hæmorrhages may occur in other parts than those referred to, and are of great importance in clearing up the diagnosis in doubtful cases—for

example, in the period before the occurrence of subperiosteal or gum changes. If a child is languid, anæmic, and manifestly ill, but shows no definite objective signs of disease, the presence of blood in the urine, or in the motions, or the appearance of a purpuric eruption, may at once suggest the nature of the case, and lead to a careful inquiry as to the diet. These bleedings are not of any marked severity in the early stages of the disease; but in the later there may be hæmorrhage in the lungs, or the pleura, or the bowel, which adds greatly to the gravity of the affection.

Cardiac weakness is often present out of proportion to the degree of anæmia, and a fatal attack of syncope is not unknown. The temperature is not, as a rule, raised; but if the periosteal hæmorrhages are extensive, with great pain from the tension in the affected limbs, it may reach 101° F. or 102° F. When persistent pyrexia is present, I have usually found that this was dependent on some complication, such as bronchitis or pneumonia. Digestive disturbances, as manifested by vomiting and diarrhœa, are frequently present, and these are of course more marked if the patient is also the subject of rickets.

Amongst the rarer varieties of scurvy are to be classed certain cases of a chronic, relapsing, or atrophic type. The subjects of this condition are usually emaciated and stunted in appearance. They are listless and irritable, and manifest an extreme dislike to standing or walking. There is general tenderness, and on examination this will be found to be situated chiefly in the bones of the extremities. In advanced cases the long bones are atrophied, and the result of this atrophy is seen in the tendency to spontaneous fracture, or fracture from very slight causes. Such fractures show very little tendency to heal, and the union is seldom of a firm character so long as the disease persists. The hæmorrhagic tendency is present, as in other forms of scurvy, but takes the form of slight oozing rather than extensive extravasation. In connection with the long bones this may be sufficient to detach the periosteum from the shaft and interfere with the nutritive process to such an extent that atrophy results, but it does not lead to prominent or palpable swellings. In some cases the disease is characterised by relapses—even when proper treatment has been persisted in for some time.

The prognosis in this chronic type of scurvy is not good, because, although the more definitely scorbutic signs subside under a prolonged course of treatment, the child usually remains emaciated, weak, and stunted in growth.

The Diagnosis and the Differential Diagnosis.

In a well-marked case of scurvy, the diagnosis is, as a rule, comparatively easy, as the clinical picture is sharply defined; but in the early stages, and more particularly when the case is complicated with rickets, the diagnosis may present considerable difficulty. In forming a diagnosis, attention is to be directed to the nature of the diet, the history of the illness, the signs present on examination, and finally the result of the therapeutic test.

The diet will usually be found to have been markedly deficient in fresh animal or vegetable food. Disorders of digestion or other causes have led to the employment of some form of altered milk or proprietary food on which the child seemed to thrive up to a certain point, and then symptoms of illness appeared. The true history of the diet is not always easily obtained, and it is important in the case of infants to get a detailed account of the feeding from the time of birth. Even those mothers who are most devoted to patent food-stuffs have a lingering belief in the efficacy of fresh cow's milk in illness, and this will often be stated to be the infant's sole food, although it may have been used only for a few days. On the other hand, the statement that the baby has been brought up on a certain proprietary article of food may turn out to mean, on further investigation, that a few spoonfuls of this food have been added to a pint or a pint and a half of cow's milk in the day—a diet which may be open to criticism, but which is not likely to induce scurvy. In the case of children under two years of age, the absence from the dietary of fresh milk, fruit, and vegetables will suggest the possibility of scurvy. In the case of older children it will usually be found that scurvy has supervened on the too prolonged use of some special dietary in which the antiscorbutic element is deficient, or that the child has an aversion to fruit, vegetables, and other antiscorbutic foods, and has not partaken of them.

The gradual onset of the illness is in marked contrast to the sudden development of the special

symptoms for which advice is usually sought. Inquiry will elicit the fact that the patient has been out of sorts for some time, as manifested by increasing pallor, listlessness, irritability, restlessness at night, general tenderness when bathed or carried about, and disinclination to walk or move the limbs. In the majority of cases a localised or tender swelling appears in connection with one or more of the long bones, and the nature and situation of the swelling are usually sufficiently characteristic to allow of an exact diagnosis being made. If any doubt exists as to the contents of the tumour, an exploratory puncture may be made with an aspirating needle, when the withdrawal of blood-stained fluid and the detection of the bare shaft of the bone will prove conclusive. The sudden onset and extreme tenderness of these blood-swellings, their localisation in the course of the shaft of the bone, the separation of the epiphysis, the freedom of the neighbouring joints, and the presence of *pseudo*-paralysis and oedema in the limb affected, are points which can usually be made out in well-marked cases. Spongy swelling, bleeding, and ulceration of the gums, most marked around the teeth which have been cut, are important signs. Blood in the urine, intermittently or continuously, may be the sole objective sign of scurvy for some time. Oedema of the upper eyelid, with blood-staining along the margin of the orbit appearing later on, or slight melæna, or epistaxis, may suggest the nature of the disease.

Finally, the therapeutic test is a most important aid in the diagnosis. A change of diet, namely, the substitution of antiscorbutic foods for the tinned, dried, or otherwise altered food materials previously employed, will lead to the amelioration of the scorbutic signs and symptoms. This improvement is usually rapid, and a distinct alteration can be observed within a few days.

(To be continued.)

The Creasote Treatment in Childhood.—Dr. Hock says, "In children the creasote treatment has a fruitful field in other than pronounced lung diseases; thus it has been found very useful in the catarrhs that so frequently remain after measles, which so frequently prepare the ground for tubercular infection. Where creasote is indicated, the author now employs creosotol."

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CLINICAL LECTURES ON URINE.

Delivered at University College Hospital by

J. ROSE BRADFORD, M.D., F.R.S., F.R.C.P.,

Physician to University College Hospital.

VI.

I POINTED out to you last time that the amount of urea that patients pass was largely dependent on diet, and that a patient may pass a small amount of urea and yet that this may not signify because it may be due to a limited diet. Further, that a patient may pass only a normal amount of urea,—thirty grammes,—and yet there may be extensive wasting going on, and the deficient urea excretion is dependent on the patient taking little or no food. There is only one class of disease where it is really important to determine accurately the urea excretion, and that is in certain renal diseases, more particularly Bright's disease, and the diseases where the kidney substance is atrophied, as for instance in cases of so-called surgical kidney, and in cases of hydronephrosis and cystic kidneys. The current text-book teaching on the subject is that a diminution in the excretion of urea in renal disease is very serious and betokens the onset of uræmia, and of course no doubt to a certain limited extent this is true; but I can only repeat that if you have a patient with renal disease who is only passing fifteen to twenty grammes of urea, you must remember that there are many causes to account for that, quite apart from the small urea excretion being due to any imminence of the onset of uræmia. A patient with Bright's disease eats very little, also he is often very sick, and hence they really assimilate still less than they eat, and then a patient with Bright's disease probably has a considerable amount of albuminuria; all these three things—diminished food, the repeated vomitings, and the albuminuria—will very largely tend to diminish the nitrogenous metabolism of the body. Then in addition the patient with Bright's disease may have profuse diarrhœa, and when there is profuse diarrhœa there is of course a very considerable loss of proteid matter in the intestinal flux. And lastly, a patient with Bright's disease is very often dropsical, and when you remember that the percentage amount of urea in dropsical exudations is as great as it is in the

blood, it is often impossible for the patient with Bright's disease to pass anything like the normal quantity of urea, perfectly impossible, so that you must not jump to too hasty conclusions because the quantity of urea is diminished in certain forms of Bright's disease. Uræmia must not be considered imminent simply because there is a scanty urea excretion. But there is no doubt that a permanent scanty excretion of urea is a matter of some gravity. In certain other diseases, more particularly in cases of so-called surgical kidney, where the kidney lesions supervene on the top of strictures of the urethra or enlarged prostate and various other impediments to the exit of urine, in such cases you may often gain valuable information as to the state of kidney by examining the urea excretion, and in these cases the percentage of urea may be very low; instead of 2 per cent. there may be only 1 per cent. or even less. The same remark applies to some cases of hydronephrosis; in uterine cancer, for instance, pressure on the ureters will cause hydronephrosis, and the long-continued excretion of a dilute urine containing a low percentage of urea suggests the presence of considerable structural alterations in the kidney, and this is more especially characteristic of surgical kidney and of hydronephrosis. These conditions are liable to be overlooked, the symptoms are often not prominent, and hence the percentage of urea in these cases may be a useful physical sign if looked for. If you are going to determine the amount of urea in the urine, it is waste of time to examine a sample; you must mix up the whole twenty-four hours' urine, otherwise you may have the most fallacious results.

There are certain diseases in which the excretion of urea is very greatly increased, more particularly, of course in diabetes. This is a disease in which the excretion of urea is much increased, and the increase is mainly due to the increased appetite, and to the fact that the diet is generally exclusively an animal one. Pathologists have supposed that the increased excretion of urea in diabetes is dependent on the disintegration of the proteids of the body. Of course it is to a certain extent so, for these patients waste largely; but in many cases the increase of urea is due to the large appetite of these patients. It has also been suggested that in some cases diabetes is a disease of the muscles, the muscles breaking up into a carbohydrate

moiety which is excreted as sugar, and a nitrogenous moiety excreted as urea.

In diabetes insipidus the amount of urea is rather large, but the percentage is very low. Patients with diabetes insipidus may pass an increased amount of urea, but the percentage is always very low, since it is not unusual for these patients to pass some twenty pints of urine. There is another condition which is rather obscure, and I should not like to say that it is definitely established, but it has been thought probable by many, and asserted positively by some, that in the true granular kidney there is an increased excretion of urea in the early stages of the malady; it is one of those things that is very difficult to prove. It is known that the initial symptoms of patients with granular kidneys are sometimes not renal, they do not come with obvious renal symptoms such as headache, vomiting, &c., they come with general weakness and emaciation, they may even have a fair appetite, of course later on that fails; but in the early stages of the disease or in certain periods of the disease, patients with granular kidney suffer more from general nutritional disorders, loss of strength and wasting, and along with these it is asserted that there is an increased excretion of urea. It is, however, quite certain that patients with advanced, I mean very advanced renal disease, who only have a few weeks or months to live, sometimes pass large quantities of urea. I remember myself a case very well in which I carried out a complete series of investigations on the nitrogenous metabolism, and that patient passed for weeks and weeks a daily average of forty grammes of urea. So that although, broadly speaking, in renal disease the urea excretion may be diminished, yet it is not really definitely shown that the diminution in the excretion of the urea is always due to impaired excretory activity of the kidney. It is often due to want of food, and to rejection of food, and to albuminuria and so forth, and certainly in some advanced cases of renal disease a patient may pass quite large quantities of urea.

Urea is of very little clinical importance apart from these facts. There is, however, one further thing to note, and that is the diminution or disappearance of the urea in the urine in certain grave diseases, and more especially in acute yellow atrophy of the liver. You may go many years without seeing that disease, although we have had

several cases here; but it is not seen very often, and some physicians absolutely refuse to recognise it. Acute yellow atrophy is a disease (of course I am giving you a cut-and-dried statement) in which the liver cells undergo degeneration *en masse*, and great areas undergo necrotic changes; it is so rare that some physicians do not believe in it, but I think there can be no doubt about the existence of the disease. But there is another condition which resembles it. When I was a house physician in this hospital, I saw several cases of phosphorus poisoning, and phosphorus poisoning produces very similar changes in the liver to acute yellow atrophy, and phosphorus poisoning is sometimes not detected unless you inquire particularly for the possible poisoning. A former student, when taking a *locum tenens*, had a curious example of this. Some people had got up private theatricals, and there was a ghost in these theatricals, and the girl who took the ghost's part covered her face and arms with some phosphorus paste; this was all forgotten after it was done, but some time afterwards this girl began to get very ill, and she died of phosphorus poisoning, and it was only discovered by close questioning how this phosphorus poisoning had been produced, and if she had not been examined so closely and accurately that case would have been undoubtedly put down to acute yellow atrophy of the liver. You will also see cases of women with a history of abortion followed by acute yellow atrophy. Many of them are connected with illegitimate child-bearing, and it has been suggested that this acute yellow atrophy in connection with the abortion is sometimes due in these cases to phosphorus poisoning. In various grave diseases of the structure of the liver, more particularly in cirrhosis and obstruction of the bile-ducts, you get what the Germans call secondary acute yellow atrophy; that is to say, after the long continuance of the cirrhotic changes or long-continued obstruction of the bile-ducts, the liver cells undergo a somewhat similar change to that seen in acute yellow atrophy, and the patients suffer from cholæmia, that is to say a condition of coma due to a toxic agent, and liable to occur as a terminal phenomenon in such cases of liver disease. This secondary acute yellow atrophy is by no means uncommon, the primary atrophy and the yellow atrophy from phosphorus poisoning are uncommon. The last is uncommon now because amorphous phos-

phorus is more used in the manufacture of matches. In acute yellow atrophy the urea excreted diminishes greatly, and it may disappear altogether. Leucin and tyrosin occur in the urine. This fact is often regarded as evidence that the liver forms the urea, because in the condition in which the liver practically becomes non-existent leucin and tyrosin appear in the urine instead of urea; that to my mind is very slight evidence that the liver forms urea. In phosphorus poisoning it was said that leucin and tyrosin did not appear in the urine, but that is now known to be incorrect; in cases of phosphorus poisoning leucin and tyrosin may be present in the urine, and the urea is certainly greatly diminished. Similarly in those cases of so-called secondary acute yellow atrophy arising in the course of hepatic cirrhosis, &c., leucin and tyrosin may appear in the urine.

Urea disappears or undergoes diminution in acute yellow atrophy, in phosphorus poisoning, and in the diseases in which the liver substance is extensively destroyed, and in these conditions lactates appear in the urine. This is a fact of some interest, and is similar to what is known to occur experimentally after the extirpation of the liver.

As regards the qualitative detection of urea, the best thing to do is to concentrate the urine and obtain the nitrate or oxalate of urea; if you want a quantitative test, everyone uses the hypobromite test. In doing that test, first of all it is necessary to work with bromine tubes and caustic soda; you cannot work with hypobromite solution, which, if it is made and kept, undergoes changes; you must make your hypobromite solution at the moment you do the test. To do the test accurately, you must measure the gas, and you must not trust to those graduated instruments for the percentages, because they are frequently wrong. When tilting the urine into the hypobromite it must be done very gradually; if you do it all at once the whole of the urine will not be decomposed. The great point, therefore, is to remember that it must be done gradually, you must be very careful that you let the hypobromite cool when you mix the bromine tube and the caustic soda; you cannot be too careful in letting it cool, otherwise there will be an error in the result. The hypobromite test is a very fairly accurate method, although it is about 8 per cent. wrong, but still, nevertheless, it is a fairly accurate method. For one thing, this 8 per

cent. error is constant, but if there be sugar in the urine this 8 per cent. error is not present; and the fact remains that sugar in the urine allows the whole of the urea to become decomposed. Hypobromite decomposes other nitrogenous bodies besides urea; it decomposes creatinin, and the various nitrogenous bodies that are present, but creatinin is the most abundant of these other substances. If you want to conduct investigations on the nitrogenous metabolism, the total amount of nitrogen in the urine must be determined, but that is not generally done. Clinically, it is usually sufficient to determine the amount of urea, but you must determine the amount of nitrogen if you wish to obtain really accurate results; Kjeldhal's method is the one most suitable for the purpose.

As regards the other nitrogenous bodies in the urine, creatinin we have considered, xanthin and hypoxanthin are of practically no clinical importance; xanthin may perhaps rarely occur clinically in large amounts, but, however, they are both pathological curiosities.

We will pass on to a very much more important subject, viz. the presence of carbohydrates in the urine. It is well to call this condition not diabetes but glycosuria; the two names are not synonymous, you may get diabetes without glycosuria, you may have glycosuria without diabetes. In a certain stage of some cases of diabetes there may be no sugar, and you can have a patient with glycosuria without the patient suffering from diabetes. The first question as regards glycosuria is the same as with regard to albuminuria, that is whether there is any sugar in the urine in health. There has been an immense amount of controversy on this subject; but it is one of those things that is of no great importance from our point of view, clinically, because whether there be sugar in the urine or not, everybody is agreed that this trace of sugar, if present, is of no pathological significance. So that to all intent and purpose with the ordinary sugar tests, the urine normally is free from sugar, but if you ask the question scientifically there is probably sugar in the urine; you have to concentrate large quantities of urine to get the result, or to use very delicate tests. Quite apart from this question there is no doubt that the normal urine contains reducing substances, and as regards these reducing substances which may be present I have mentioned uric acid, and I have mentioned creatinin; in

addition to these you have a carbohydrate acid that is known as glycuronic acid, which is probably always present in traces as far as we know, and which in certain conditions is very much increased, and this acid is a reducing body, although it is not sugar. The amount of glycuronic acid is increased after the administration of camphor and other drugs, and its presence in the urine was first determined after the administration of this body and of chloral. The question as to whether the normal urine contains sugar, narrows itself down to whether the urine contains substances that will ferment, or will crystallise with phenyl-hydrazin. It is an experiment about thirty years old, that if you concentrate the urine you can get out of it a reducing sugar, but it is really of no great practical importance. Putting all that aside, there is unfortunately, because it complicates the subject, there is unfortunately a condition somewhat similar to so-called physiological albuminuria; that is to say, there are certainly patients who pass appreciable quantities of sugar in their urine, these people being as far as can be determined healthy. I may as well say at once, to avoid any misconception, that the more strict insurance offices will not accept lives on the usual terms when the urine contains sugar, even if the people are apparently in perfect health. So that most observers are inclined to look on glycosuria as more serious than albuminuria, but whether that rests on any sound evidence or not is another matter. As regards those persons who pass a small quantity of sugar in health, you see it principally under two conditions. First of all, after diets rich in carbohydrates, there are certain people who will pass a small quantity of sugar; after eating largely of carbohydrates, they pass no increased quantity of urine, and they are apparently healthy. There is another group of cases in which a person passes sugar in his urine after moderately severe exercise. I have only seen one case; the patient was a student here, who found that he had sugar in his urine after a ten mile walk. It was tested quite accidentally; he was going to have his life insured. It is very difficult to say whether these people who pass sugar in their urine after a meal or after exercise are slight cases of diabetes or whether they are not; they are not perfectly sound of course, there is no doubt about that, but whether they are people who will die of diabetes

or whether they will live as long as anyone else is really a question that one cannot answer; the fact remains that you cannot pass them as healthy people for insurance purposes.

The next point is that you must not say that a person has got sugar in the urine simply from having noticed a reducing body in their urine. You must never say that a person has sugar in the urine simply on the basis of the copper reduction test; you must always ferment it, or use some other equally good test. The fermentation test is a good one for the purpose of a control test.

Pathological glycosuria is a large subject. First of all, one would say that there are at least three types of diabetics. First, there is a type every one is familiar with, the urine is increased in quantity and contains sugar; it not only contains sugar, but it is greatly increased in quantity. In the second place is a class of cases in which the urine contains sugar, and it is not increased in quantity, or rather not largely increased in quantity. In both these classes the specific gravity of the urine is raised, so that it is probably somewhere between 1030 and 1050. The third class of cases is that in which sugar is present in the urine, but the specific gravity is lower than normal. You may have sugar in the urine with specific gravity of 1010. In the first group of cases the urine may contain large quantities of sugar, and the condition is often serious; the colour of the urine is altered, and it often has a peculiar greenish opalescent colour. In the second class the colour is not profoundly altered. In the third class of case the urine is pale coloured, as would be expected.

In diabetes mellitus the sugar present is dextrose, and this is a point of some pathological interest. As regards the amount, you may have up to a couple of pounds in the twenty-four hours; the most I have seen has been one and a half pounds in the twenty-four hours; it is not at all uncommon for the patient to pass one pound of sugar in the urine for months and months, so that the amounts of sugar that are lost in this way are very considerable. Then the percentage of sugar is anything up to about 10 per cent. The amount of sugar in the blood normally is in the second place of decimals; it is usually estimated at '09 per cent., that is supposed to be about the maximum, the minimum is '05 per cent., so that there is roughly speaking about four or five times as much sugar in the blood

as there is urea, because the percentage of the urea in the blood is about '02 per cent. If the percentage of sugar in the blood rises even slightly you get sugar in the urine, and if it rises to '3 per cent. you get marked quantities of sugar in the urine.

We have considered the types of urine, and we have also considered the amounts of sugar present in the urine in diabetes mellitus; and now we will consider the other conditions in which sugar is found in the urine; larger quantities are found of course in diabetes than in any other condition, and the first question is whether you ever have sugar in the urine apart from diabetes. That was more easily answered a few years ago than now; one used to teach that you got sugar in the urine in a large number of cases: first of all, after injuries of the head; secondly, after a number of drugs of which opium, morphia, chloral, and chloroform were the principal; thirdly, after, or as the accompaniment of certain inflammatory diseases of the brain, more particularly meningitis; and fourthly, in any conditions such as cerebral hæmorrhage, particularly infra-tentorial, either in the cerebellum or in the pons; and fifthly, in tumours of the brain. As I say, a few years ago one said this dogmatically, now one has a certain amount of hesitation about it; it would be better to say that in all these conditions we have reducing substances in the urine, but we are doubtful whether in all cases it is sugar, after opium and chloral it is doubtful whether it is not glycuronic acid. People have relied on the copper reduction to show the presence of sugar. As regards blows on the head there the evidence is a little more clear, but no one yet has re-investigated the matter as regards the presence of glycuronic acid.

I need only say that it is a matter of traces of these substances. I can quote one case to illustrate this point. A patient in this hospital complained of noises in the ears, he had got a little deaf, and then he complained of a stiff neck, then he found that he could not pass his water properly. He still complained of the stiff neck, he was sick, and it was supposed that he had been poisoned with some ptomaine; as he was still sick and did not pass his urine he was brought into this hospital. He became comatose in the ambulance, and died a few hours after admission. That man had traces of sugar in the urine. He had an aneurysm of the

basilar artery which had ruptured. It is an instance of sugar in the urine from a local increase of pressure on the medulla, and it is one of the best instances that I have seen.

The Relief of the Immediate and Remote Effects of Phimosis by Circumcision.

BY

CAMPBELL WILLIAMS, F.R.C.S.

AMONGST the minor operations of surgery, apart from dentistry, circumcision for the relief of phimosis is perchance that which is most frequently performed. Excluding those instances in which amputation of the prepuce is resorted to as a religious rite, there nevertheless remain a residue of cases which from a numerical stand-point constitute it as the most common surgical interference to the male body. The troubles engendered by phimosis may be met with from birth up to green old age. The wisdom of the Mosaic law, whether it be viewed as a moral, sanitary, or curative procedure, seems to be incontestable, and to admit of no controversy. The bulk of male children are born "phimotic," but the degree and quality as well as the effects produced thereby vary considerably. In considering these effects, one must divide them into "immediate" and "remote." Some of them seem to be directly dependent upon the tension exerted upon the urethra, and are intimately associated with the amount of force that is expended to overcome the impediment to free micturition, and may be termed the results of "obstruction or constriction." The remainder, excluding paraphimosis, which has a mechanical origin, may be traced directly or indirectly to the presence of retained subpreputial secretion. By means of the following table I will endeavour to group these effects.

I. THE RESULTS OF OBSTRUCTION OR CONSTRICTION.

a. Difficult or painful micturition. Retention.

b. Herniæ: a. Production.

β. Maintenance.

c. Prolapsus ani. Piles (aggravation).

d. Vesical hypertrophy.

e. Prostatitis. Prostatic hypertrophy.

f. Renal disease.

} Due to straining.

2. THE RESULTS OF RETAINED SMEGMA.

1. Balanitis. Balanoposthitis.

2. Incontinence of urine.

3. Masturbation.

4. Fits and reflex neuroses.

5. Epithelioma.

3. THE RESULT OF CONSTRICTION (INDUCED). Paraphimosis.

In Group 1, the cause may be found in the size of the urinary outlet of the prepuce. This may be entirely absent, so that surgical interference may be required shortly after birth so as to allow of evacuation of the urine. Frequently the orifice is only the size of a pin-hole, but, on the other hand, it may be large enough to freely expose the meatus urinarius, and seemingly not likely to cause impediment to the free passage of urine. Under the latter circumstance when obstruction exists it appears to be due to "*the constriction exerted by a tight and adherent prepuce on that portion of the urethra which is situated within the glans penis.*" The act of micturition may not only be difficult, but painful, the water either dribbling away in small quantities or being ejected with increased force in a diminished-sized stream. When painful the child cries each time it has to urinate, and consequently refrains from doing so until the bladder becomes fully distended. If the condition of phimosis be not rectified, the increased efforts of the vesical muscles to overcome the penile resistance, tends to the production of hypertrophy of the viscus. Many a mother experiences anxiety because her child has not passed its water with that frequency which is usual at its age, the cause of this being pain from an eczematous state of the prepuce, and not from an intra-urethral irritability. The latter seems to be due to distension of the urethra.

As regards herniæ, I believe, though it is difficult to prove, that the "straining" during micturition does produce them. It certainly tends, like flatulent distension of the intestines, to maintain

and increase a rupture. It is well known that inguinal hernia is much more common in baby boys than is a similar condition into the canal of Nuck in baby girls. In those infants who are the subjects of this complaint, the co-existence of phimosis with a "minute aperture" in the prepuce is of such frequent occurrence that it suggests more than coincidence in the dual combination. For ruptures are present, in my experience, most often with the higher degrees of phimosis, in which, as one finds during operation, the prepuce is closely adherent to the glans penis. It would seem as if the liability to hernia went hand in hand with the amount of resistance offered to the egress of the urine. That the presence of this condition contributes not only to the maintenance, but to the increase in size of "existing" herniæ, is without doubt.

Although one cannot say that ruptures when treated with properly fitting trusses will not get well in unrelieved cases of phimosis, nevertheless it is fairly safe to affirm that this end would be attained more speedily and certainly if the patient were to be circumcised. In addition it may be necessary to do a meatotomy in order to enlarge a preternaturally small urethral orifice.

Prolapsus ani is primarily due to a lax and enfeebled condition of the muscles of that region. Any cause that produces straining tends to occasion extrusion, and if phimosis exists, it should be remedied with a view to ameliorating the rectal trouble. In cases of hæmorrhoids the same deleterious influence may aggravate the anal mischief.

In Group 2 we have to deal with conditions resulting from the irritating effects of retained smegma. In infants this appears as firm, yellow-white masses lying in the furrow behind the glans penis.

Balanitis or balanoposthitis (inflammation of the surfaces of the glans and the inner prepuce) are liable to be met with so long as a state of phimosis exists. It is set up by the irritating action on the tissues of the decomposed fatty secretion. The intensity may suffice to produce a copious purulent flow. In the adult this is sometimes mistaken for gonorrhœa, and results from uncleanness. Many people are unable to retract the prepuce, and are therefore debarred from the necessary daily ablution with soap and water which in these cases is so urgently required owing to excessive secretion.

Sometimes the condition is of a gouty origin, and may be very intractable to treatment, even when free access can be had to the site of the inflammation and scrupulous cleanliness is employed. In children repeated attacks of balanoposthitis lead to thickening and induration of the prepuce. This change in the tissues is very noticeable during circumcision, the arteries being markedly larger than usual, and the hæmorrhage more profuse. From a moral and hygienic point of view one cannot too strongly urge the removal of the phimotic prepuce as a prophylactic measure against masturbation. Children are apt to pull and rub the penis in their efforts to obtain relief from the irritation caused by the smegma. Presumably at a very early age they derive pleasurable sensations therefrom, and thus initiate themselves into a habit pernicious alike to their mental and bodily welfare. In many cases this practice can be broken by a timely operation, the necessity for which is too frequently ignored or overlooked until the custom is well established, or the condition of paraphimosis draws attention to the child's sensual proclivities.

Phimosis is *one* amongst the many causes that induces nocturnal incontinence of urine, apparently from reflex stimulation of the lumbar centres by an irritable glans penis. In many cases the trouble may either be alleviated or cured by circumcision. But it is well to warn the parents that the cessation of the bed-wetting is more often a gradual than an immediate process. Should no improvement follow the operation one must cast around for some additional cause that co-existed and still remains. Though circumcision may have failed to correct the condition for which it was directly undertaken, one still has the satisfaction of knowing that should the child reach manhood's estate, that *it* has at least "*relieved him of the dangers that may attend a soft, moist, and easily inoculated prepuce.*" If parents could but realise the possible "*remote*" troubles that may befall a neglected phimosis, one would seldom meet with subpreputial chancre or its sequel phagedæna. Many a victim of the ravages of syphilis can trace his impaired health to his readily lacerated foreskin. I say this advisedly, for I know of two instances in which the circumcised have escaped infection, whilst the phimotic have contracted disease *a few hours afterwards* from the same woman. Squamous epithelioma of the penis occurs most frequently in those who

possess a long, tight, and irretractable prepuce. The pre-cancerous stage is the appearance of warts, born and bred presumably by the irritation of retained and decomposing smegma. Passing allusion has already been made to the rôle enacted by phimosis in the production of vesical hypertrophy. The increased *vis à tergo*, requisite to overcome the penile resistance, leads to a *compensatory* overgrowth of the bladder muscles. If the obstruction be removed in early life the developing viscus responds readily to the altered circumstances, and the condition apparently undergoes resolution. But in those cases which are allowed to go unrelieved through adult life, the sequelæ incidental to obstruction eventually appear. Sooner or later an atonic condition of the organ heralds the advent of vesical dilatation. This is probably due to a compound effect in which the prostate plays its part. As is well known, stricture of any portion of the urethra acts as a mechanical factor in the production of *prostatitis* and *hypertrophy of the gland*. Nor does the evil effect of pressure cease with the bladder and prostate, but by *spreading upwards* to the *pelvis of the kidneys* is a fertile source of *renal* disease.

Amputation of the prepuce has been practised with a view of removing a possible exciting source of irritation in children who are the subjects of *epileptic* fits, and also in cases of *joint* troubles that have been considered to be "reflexly neurotic."

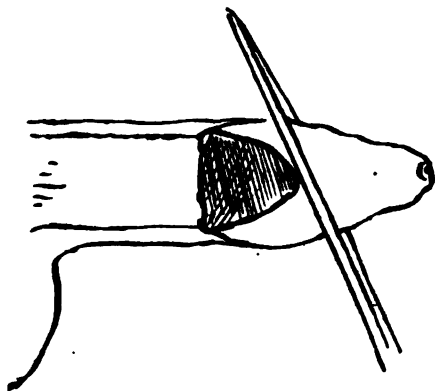
It will already have been gathered from this article that I advocate the relief of all degrees of phimosis by circumcision. I do not believe in half measures, and therefore condemn stretching and retraction, or slitting up the prepuce. Stretching is frequently more than unsatisfactory, for not only does it set up inflammation, swelling, and discharge, but when these symptoms subside the part is apt to become *even more contracted than it was naturally*, and eventually circumcision has to be performed, either to rectify the original trouble, or to relieve that which supervenes from dilatation. It may be difficult to obtain the consent or convince the parents of the necessity for this trifling operation, for the word is often a "bogey" to them. In their anxiety to shield the child from imaginary dangers, they unwittingly wrong him, in that they do not permit what can only be for his ultimate good. Under these circumstances it seems to be the duty of the practitioner, when he considers

the operation necessary, or even advisable, to impress upon them the benefits that it will confer upon the boy, not only in the immediate but also in the remote future. A lucid explanation of the gains—moral, sanitary, and prophylactic—that will accrue to the patient after its performance will usually succeed in turning the balance of parental indecision.

I do not employ the same mode and technique in circumcising the infant and the adult, but this maxim holds good with both: "Do not remove too much or yet too little skin." There is nothing more unsightly than the redundant "blob" of skin that one sometimes sees left from an endeavour to avoid cutting the *frænum*. Again, it is hard to believe, unless one has seen it, that though the *dermal* portion of the prepuce has been *removed*, the *mucous* has been left *in situ*, *untouched* and still adherent to the glans. The following method seems to answer best in children and gives a neat, effective, and quick result, raw surfaces and healing by granulation being avoided. The instruments required comprise a thin pair of sinus forceps, a pair of sharp, flat, blunt-pointed scissors, a silver probe, two pairs of Spencer Wells forceps, and a needle armed with horse-hair.

The patient being anæsthetised, the part is well cleaned, first with soap and water, and then with some 1 in 20 carbolic lotion. The prepuce is now retracted with the fingers, but sometimes this is not practicable owing to the small orifice, or to the presence of adhesions between it and the glans penis. In the latter case they may be broken down, and the surfaces separated by introducing a silver probe and forcibly sweeping it around. If it is the skin that forms the barrier to retraction, it should be first excised, and the mucous membrane dealt with subsequently. When the prepuce admits of the exposure of the glans, it is peeled backwards, any slight adhesions being broken down by the thumb nails, until that body is entirely exposed. The smegma having been wiped away with boracic lint pads, and the part well cleaned, care being taken to thoroughly free the mucous membrane should it be adherent to the corona of the glans, the prepuce is now replaced and drawn *very slightly forwards*. This must not be overdone for fear of removing too much skin. The sinus forceps, taken in the left hand, are used to firmly grasp the prepuce, *not vertically*,

but with a slight slant from above downwards, so that a little projecting tongue of skin will be left below.



Before proceeding further, feel and make sure that the *tip* of the glans penis is not *included* between the blades of the forceps. The scissors are taken in the right hand, and with one snip, cutting closely along the upper surface of the forceps, so that the glans is protected by the intervening metal blades, the foreskin is removed. The blunt point of the scissors is next introduced *by the side* of the frænum, and the mucous membrane is divided in a circular fashion by following round the furrow until the opposite frænal aspect is reached. A frill of mucous membrane from $\frac{1}{8}$ to $\frac{3}{16}$ of an inch in depth is left. Bleeding vessels are now searched for and twisted with Wells forceps. It is only in elder children and adults that one may occasionally need to employ ligatures. Torsion usually suffices. The little tongue of *skin* on the under surface is now adjusted and fixed with a horsehair stitch to the mucous membrane of the frænum. Four more stitches suffice to join the remainder of the cut surfaces. It is advisable to pass the needle as close to the free edges of the tissues as is compatible with efficiency, as then the horsehair works out of its own accord and saves the surgeon the trouble that their removal entails in a frightened and struggling child.

A narrow strip of buttercloth spread with boracic ointment is wound around the cut to prevent the sticking of the little bandage of mercurio-zinc-cyanide gauze with which the wound is dressed. The free ends of the bandage are fixed to the child's abdomen by collodion. A pad of Gamgee

tissue, with a hole cut in it for the penis, is put on as a guard.

Particular attention should be paid to the arrest of hæmorrhage, for sometimes children are in a state of syncope from the anæsthetic, and owing to an enfeebled heart's action do not bleed towards the end of the operation, but *on coming to* may do so to such an extent as to necessitate opening up the wound to secure the bleeding point, or a clot of blood may form beneath the skin, and breaking down may require removal before healing takes place.

In the adult I prefer to start by cutting through the dorsal surface of the prepuce, and then proceeding on either side towards the frænum. With these a lasting dressing of blue wool and collodion is employed, care being taken to exclude the possibility of external infection of the wound by sealing up the ends with collodion. By this means union by "first intention" should be obtained.

NEUROTIC SPANOPNŒA and TACHYPNŒA.

RETARDED respiration and accelerated respiration of a neurotic nature have lately been made the subjects of investigation by Strübing ('Zeitschrift für klinische Medicin,' xxx, 1 and 2; 'Centralblatt für innere Medicin,' July 17th, 1897). He remarks on the complicated action of the cerebrum, not yet explicable, upon involuntary respiration and upon the development of those nervous forms of respiration which, being of reflex origin, are to be referred to irritation of peripheral nerves. In the case of a neurosis, with changed conditions of excitability, something besides the ordinary physiological laws must come into play—abnormal excitability of the centres, hyperæsthesia of the peripheral nerves, and corresponding pathological reflex processes—as in hysterical cough, in certain forms of spasm of the glottis, and in *pseudo*-asthma. By means of clinical histories he illustrates the chief types of the nervous disturbances of respiration which are brought about by hysteria, by neurasthenia, and by organic diseases of the nervous system that increase irritability. If the lungs and heart are healthy, he says, the dominant and never-failing symptom is *besoin de respirer* occurring in paroxysms; the breathing is retarded

or hastened during the seizure, the relation of inspiration to expiration may be deranged, with severe sensations of anxiety, occasionally ushered in by an aura; there may be paræsthesia of various kinds in the larynx, the neck, and the thorax, and, if a nasal affection is the cause of the attacks, there may be manifestations of aprosexia.

If respiration is delayed, the respiratory movements are very forcible, the auxiliary muscles being called into play, even to the extent of dyspnœa, and the agony of the situation is depicted in the patient's face. In one of two cases of which the histories are given there was a neurosis of the superior laryngeal nerve, excessive irritability of the fibres that inhibit respiration. That this was directly caused by a preceding laryngitis the author thinks less probable than that it was due to the irritation of a uterine affection, for the cure of the latter was quickly followed by recovery. The act of swallowing sufficed to cut short the paroxysm. In the other case, analogous but not so severe, irritation of the trigeminus caused constrained breathing; chronic rhinitis was present, and indigestion brought on the attack.

Opposed to this inhibitory neurosis, spanopnœa is tachypnœa occurring in paroxysms, but not accompanied by a feeling of dyspnœa, except in its pronounced forms. Whether this is due to depression of the inhibitory mechanism or to irritation of the accelerator nerves of respiration cannot yet be determined, but that pathological irritation of the terminal expansions of distant nerves may give rise to such a tachypnœa seems to be exemplified by one of the cases.—*New York Medical Journal*, September 4th, 1897.

CENTRILOBULAR AND PERICELLULAR CIRRHOSIS.

Dr. J. E. ADAMI in the 'Montreal Medical Journal' concludes a long article on cirrhosis with remarks regarding these two interesting phases of hepatic disease. The centrilobular form apparently only develops as a consequence of chronic heart or lung disease associated with passive congestion of the liver and the condition of nutmeg liver. Passive congestion in general may lead to fibroid overgrowth originating around the veins, and it is around the central intra-lobular branches

of the hepatic vein that such fibroid overgrowth may at times be recognised. While sundry French observers would ascribe one form of hypertrophic cirrhosis to this central development of fibrous tissue, it is difficult to agree with them. The liver of passive congestion may be larger than normal, but congestion is not hypertrophy, and associated with any degree of centrilobular cirrhosis there is marked hypertrophy of the liver cells, while the amount of connective tissue laid down is relatively slight as compared with what may be developed in perilobular cirrhosis. It is unfortunate that this term, hypertrophy, has been introduced at all in connection with cirrhosis. This form also is incapable of being recognised clinically; it may be suspected in long-continued obstructive disease. It is in every respect one of the minor forms. Regarding pericellular or replacement cellulitis he would say, that while he is inclined to think that in portal cirrhosis there is primarily an overgrowth of connective tissue around the medium-sized branches of the portal veins, and recognise a similar deposit occurring around the bile-ducts, arteries, and branches of the hepatic veins, there is another highly important deposition or development of new connective tissue in the liver which yet remains to be discussed, one which has up to the present received too little attention, although from time to time high authorities such as Kelsch and Wannebrouck, Hamilton, Beale, and Grandmison have more or less called attention to it. Where isolated cells of a tissue or collections of cells atrophy and die there is in general an attempt to replace them, if not by cells of a similar nature then by connective tissue, or, failing this, by fluid (as in some cyst formations). A replacement-fibrosis of this nature may be localised, as after scattered necroses through the liver substance. Such fibrosis very possibly explains in part the cirrhosis occurring in malaria, and in those infectious and septic diseases in which sporadic necroses of the liver are becoming more frequently recognised. Or, on the other hand, it may be more generalised, affecting the periphery of the lobule, or, in some cases, the whole lobule. In the Pictou cattle disease this could be seen in its various stages. Pictou cattle disease is a chronic infectious disease of cattle in Nova Scotia, characterised especially by extensive cirrhosis of the liver.

In early cases, as first pointed out by Dr. Wyatt Johnston, the liver cells show various evidences of degeneration, and this stage is followed by atrophy so extensive that in advanced cases all the cells of some lobules may become unrecognisable, their place being taken by a delicate connective tissue. In other regions, short columns of the atrophied cells may be isolated and surrounded by similar connective tissue, while, as in biliary cirrhosis, there may be very numerous imperfectly formed bile canals, examples of which have been termed "reversionary" degeneration of the liver cells. Along with this there is in general singularly little small-celled infiltration. The process is essentially one of hypertrophy of the liver cells by toxic agency followed by replacement-fibrosis. It is interesting to note that in all cases where this pericellular cirrhosis is well marked the liver tends to be enlarged—hypertrophic,—or if not enlarged is not found hobnailed. There appears to be a difference in the way in which the new connective tissue is laid down. Whereas in ordinary portal cirrhosis there is a frankly inflammatory infiltration of small round cells, followed by connective-tissue growth, the new tissue is typically cicatricial, *i. e.* with full development it contracts very markedly, and so produces the hobnailed condition. In such cases the atrophy of the liver cells is secondary, and is, in the main, due to the presence of the fibrous bands. In the cases of extensive replacement-cirrhosis above referred to, there is curiously little small-celled infiltration; a loose transparent connective tissue develops which evidently is not nearly so prone to contract. Degeneration and atrophy of the liver cells may be brought about in four ways: 1. By pressure. 2. By toxic substances reaching the cells by the circulation. 3. By toxic substances reaching them along the bile capillaries. 4. By arrest of function resulting from obstruction of the bile-ducts. To the first of these we have already referred; the contraction causing the atrophy does away with the opportunity for a replacement-fibrosis. About the third possible way we know little. It is unlikely that toxins should diffuse up the bile canals in a direction contrary to the flow of bile. With regard to bacteria penetrating the liver along the bile-ducts and producing toxins, we are in doubt. We know from the researches of Welch, Flexner, and others, that pathogenic microbes are not infrequent in the gall-bladder,

and in the last two cases of hobnailed liver coming to the post-mortem room of the Royal Victoria Hospital we were not a little surprised to gain cultures of the *B. coli-communis* (the most common form to be found in the gall-bladder) from the liver juice when other glandular organs did not show this form. But, on the whole, it is for the present to be regarded as unlikely that degeneration of the liver cells and cirrhosis are induced to any considerable extent by toxic agencies passing up the bile-duct and capillaries. It would seem more probable that obstructed and perverted action of the liver cells by closure of the bile-ducts, leads to their degeneration.—*Journal of Amer. Med. Assoc.*, September 4th, 1897.

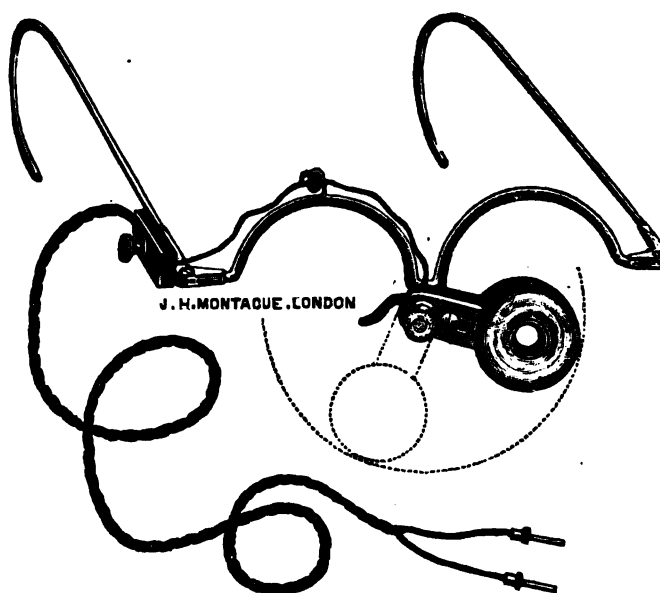
NOTES.

Pertussis.—Fröhlich states that leucocytosis usually exists in these cases, and that it is often quite marked. He especially observed it in cases examined in the third and fourth week, at the period when the paroxysms were most severe. The patients examined before and after this period showing a less leucocytosis; the augmentation of the white blood-corpuscles must have occurred with the increasing intensity of the attacks. Decrease of white corpuscles caused renewed reduction of the intensity of the paroxysms. Patients suffering from diseases which of themselves gave rise to a leucocytosis were excluded from this series.—*Jahrbuch für Kinderheilkunde*, vol. xliv, No. 1, p. 53.

Lumbar Puncture.—Babcock gives indications for this operation, and reports nineteen cases in which puncture was done. Twelve were general paralytics, two simple melancholiacs with pressure symptoms (intense headache, stupor, photophobia), and the remaining five included one case of each of the following maladies: locomotor ataxia, stuporous melancholia, organic dementia, status epilepticus, and acute delirium. A careful study of the results obtained brings out the following conclusions: 1. Lumbar puncture affords temporary relief from pressure symptoms in over 50 per cent. of cases of paresis submitted to the operation. 2. The most beneficial effects are

manifest over motor inco-ordination, *i. e.* ataxia, tremors, &c. 3. Analysis of the fluid obtained in paresis shows that it contains an inflammatory product (albumen) throughout all stages. 4. It may be of benefit in locomotor ataxia, status epilepticus, or organic cerebral disease, and deserves further trial in these cases. 5. It presents excellent diagnostic possibilities, particularly in meningeal inflammations. 6. It does not sufficiently benefit melancholia with pressure symptoms to warrant its use in this disease. 7. Re-accumulation usually occurs within from three to ten weeks, when a second or even a third puncture is indicated if patient's condition admit.—*International Medical Magazine*, May, 1897.

to avoid any unpleasant radiation of heat to the eye the back is further protected with non-conducting asbestos material. A small transformer is required for use with the alternating current, the lamp working best at about 23 volts. At present this lamp has been tried in two forms; (1) attached to a handle provided with a switch, and (2) arranged for laryngoscopic work, in which it is hinged to the bridge so as to be readily adjustable for either eye; a switch is fixed to the right side of the frame, which can be turned on or off by the slight movement of a small milled head. The advantages of this lamp consist in its lightness as compared with a laryngoscope mirror, of which it takes the place, and more especially in its ren-



A NEW SURGICAL LAMP.

THE accompanying illustration represents a new form of electric lamp made at my suggestion some time ago by Mr. J. H. Montague, of 101, New Bond Street. Having had frequent opportunities of practically testing its usefulness, I have found it very convenient in more than one application. Though originally designed for ophthalmic work, the lamp, which is one inch in diameter, is circular, flattened from before backwards, and perforated by a central aperture about a quarter of an inch in diameter; the filament completely encircles this aperture. The back of the lamp is silvered and blackened round the central hole behind, &c.,

dering the usual bracket lamp unnecessary, thus obviating the frequent adjustment of this and of the mirror which is generally required; for the lamp once fixed satisfactorily in the line of vision needs no further alteration, whatever the movements of the observer or patient. The uses to which this lamp can be put are many; the most important are: for indirect ophthalmoscopic examination (here the laryngoscope frame is very convenient, for it leaves the hands free), for laryngoscopy, and for examination of the ear and nose. It is also applicable to the urethroscope.

H. R. BELCHER HICKMAN, M.B.Oxon.

122, Harley Street.

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CLINICAL OBSERVATIONS ON SCURVY IN EARLY LIFE.

BY

G. A. SUTHERLAND, M.D.Ed., M.R.C.P.Lond.,

Physician to Paddington Green Children's Hospital.

PART III.

The Differential Diagnosis.

1. *Rickets*.—All authorities are agreed that rickets is a very frequent accompaniment of scurvy. Out of 100 collected cases of scurvy I find that rickets was definitely present in fifty-eight, was definitely absent in twenty-one, and that particulars are not mentioned in the remaining twenty-one cases. The extent of the rachitic affection may be very great, or moderate, or extremely slight. The onset of rickets may precede the scorbutic affection, or may occur simultaneously, or may come later. In some cases it would appear as if scurvy had been the exciting cause of alterations in the bones of a rachitic nature, as these have not been manifested until after the absorption of the hæmorrhagic extravasations.

In the present day the dualistic theory—that rickets and scurvy are different diseases—has been accepted in England and America, and to a more limited extent in Germany.

In a paper entitled "Barlow's Disease (Rachitis Hæmorrhagica)" Dr. Fiirst has described most fully a case of scurvy in an infant which terminated fatally. The history of the diet, the signs and symptoms during life, and the changes found at the necropsy clearly show that this was a case of scurvy. The only diagnostic point which is wanting is the result of antiscorbutic treatment, and this treatment unfortunately was not employed. Dr. Fürst, however, although he records this as a typical case of "Barlow's disease," denies that there is any proof of the scorbutic nature of this affection, and, curiously enough, he adopts the alternative title of "rachitis hæmorrhagica" for this case, although

careful examination during life and after death had failed to reveal the slightest trace of rickets. He holds that rickets may be present without this special "hæmorrhagic diathesis," or the special diathesis without rickets. The particular cachexia which develops, rachitic or hæmorrhagic, or a combination of both, is dependent on the absence from the diet of phosphate of lime, or phosphate of potash, or of both. This purely chemical theory as to these diseases will not stand the test of experience, and has long been discarded in this country.

Dr. Ashby's view of the "so-called scurvy rickets" is that "the hæmorrhagic diathesis is simply an exaggeration or an excessive form of the anæmia which is always present in severe or acute rickets." Out of twenty-five cases he has met with, in all the rachitic manifestations were well marked. This experience of Dr. Ashby's is not in accordance with that of others, for, as has been stated, rickets is by no means invariably present, and typical examples of scurvy arise quite independently of that affection. Nor does experience show that a condition of "acute or severe rickets" is necessary for the development of hæmorrhagic signs, but the so-called acuteness or severity of the rickets is really caused by the extravasation of blood, and the relief of the latter by antiscorbutic treatment is accompanied by the disappearance of the acute rachitic symptoms.

The differences between scurvy and rickets are recognisable in the ætiology, in the clinical manifestations, and in the results of treatment. As regards the ætiology, although in both affections an imperfect diet plays a very important part, the nature of the defect is different, in rickets it being largely the absence of fatty materials, while in scurvy it is the absence of fresh or "living" food. As a matter of experience it is found that both of these factors are often combined, and hence the frequent association of the two diseases. If the antirachitic material alone is wanting then rickets supervenes, and if the antiscorbutic element alone is absent, then scurvy develops.

The clinical course of the two diseases is different,

and the distinguishing features of scurvy have already been given. The leading sign, namely, hæmorrhage, is not an accompaniment of rickets *per se*, and the extensive subperiosteal hæmorrhages were quite unknown to the authorities on rickets of thirty years ago. Sir William Jenner, on seeing some of Dr. Barlow's specimens of scurvy in infants, said he had never met with such a condition in cases of rickets. It is impossible that such marked pathological changes could have been overlooked, and those who regard the extensive hæmorrhages as indicating merely an acute form of rickets have to explain how this development has taken place within recent years. Rickets is a chronic constitutional disease characterised by general feebleness, and accompanied by local manifestations in the bones (periosteal and epiphysal softening and overgrowth), in the mucous membranes (catarrh), and in the teeth (delayed dentition). Scurvy may be defined as a chronic constitutional affection characterised by progressive anæmia, lassitude, and debility, and accompanied by local hæmorrhages, affecting the bones (the periosteum and epiphyses), the gums, the skin and subcutaneous tissues, the mucous membranes, and the viscera, and producing in certain parts painful swellings and *pseudo*-paralysis. If the two diseases are combined they will of necessity act and react on each other, modifying to a certain extent the distinctive constitutional and local symptoms. Constitutionally the effect will be seen in the greater weakness and prostration and anæmia present. Locally the active growth and increased blood supply in certain structures when rickets occurs will determine the site of the hæmorrhage if scurvy be superadded. Conversely, the cessation of active growth in certain parts due to rickets will remove the tendency to hæmorrhage in those parts, and hence the frequent absence of sponginess of the gums in rickety infants in whom dentition is delayed.

The great difficulty in diagnosing between rickets and scurvy arises in connection with a class of cases to which Dr. Barlow has given the name of "borderland," that is to say, those cases in which rickets is undoubtedly present, and there are in addition some symptoms which are suggestive but not diagnostic of scurvy. A similar difficulty has long been recognised in connection with rickets and syphilis, and even yet there is no agreement

as to where rickets ends and syphilis begins, or *vice versa*. It may be at once admitted that at present it is difficult if not impossible to state exactly where rickets ends and scurvy begins. Some writers lay stress on the tenderness of the bones in rickets, while others do not regard this symptom as of special frequency. Without entering into this question more fully, I may refer to the experience of Drs. Cheadle, Eustace Smith, and Barlow, that a marked degree of tenderness is very suggestive of scurvy. If a rachitic infant is irritable and disinclined to move its limbs, cries when touched or bathed, and is somewhat anæmic, the adoption of an antiscorbutic diet will frequently lead to the disappearance of the lassitude, irritability, tenderness, and anæmia.

(2) *Syphilis*.—The diagnosis here is often extremely difficult if the physical signs only are considered. Syphilis and scurvy may be present at the same time in the same patient, and even if the syphilitic disease is not active it may so affect the scorbutic symptoms as to make them very puzzling. A painful swelling appears at the extremity of one of the long bones, which spreads from the epiphysal region along the diaphysis, under the periosteum, and leads to loss of power and finally complete *pseudo*-paralysis in the limb affected. In syphilis this is a form of epiphysitis; separation of the epiphysis may take place, and suppuration not infrequently follows. In scurvy the effusion is hæmorrhagic, and inflammatory changes and suppuration are absent. Further, in syphilis I have sometimes observed œdema and swelling of the upper eyelid, which was evidently dependent on thickening of the periosteum of the orbital plate, and which is more frequently an important sign of scurvy. On the other hand, in scurvy I have seen hæmorrhagic extravasation under the periosteum of a phalanx which simulated the much more common syphilitic dactylitis.

In distinguishing between these affections it is to be noted that this form of syphilitic bone disease occurs usually in infants under six months of age, while scurvy is rare until after that period. Mr. J. H. Morgan states that in infantile syphilis changes in the bone originate more especially in connection with the humerus and the tibia. In scurvy the lower limbs are affected much more frequently than the upper, and the lower end of the femur is in a majority of cases the part first

involved. The brawny thickening due to extravasation into and between the muscles in scurvy is not found to occur in syphilis. In syphilis the epiphysal affection is rare, in scurvy it is common. In a doubtful case of a fluctuating swelling around the epiphysis an exploratory puncture may be made through the periosteum, when the presence of either pus or blood will clear up the diagnosis. A careful examination of other parts of the body will usually afford additional information, as the occurrence of bony lesions without any other evidences of syphilis or scurvy is very rare.

(3) *Acute septic osteo-myelitis*.—This disease, like scurvy, manifests itself in connection with the epiphyses of the long bones, and more especially the epiphysis at the lower end of the femur. Intense pain, swelling, and suppuration under the periosteum and in the medulla are present, and may lead to a diagnosis of scurvy. This acute septic disease is to be distinguished by the nature of the onset, which is sudden, and often accompanied by rigors or convulsions, by the greater degree of pyrexia, by the evidences of local inflammation, and by the marked pyæmic symptoms which rapidly ensue.

(4) *Rheumatism*.—In children under two years of age a difficulty in diagnosing between scurvy and muscular rheumatism is not likely to present itself, but after that age it sometimes occurs. The patient may complain of pains in the limbs on walking, and tenderness on pressure over the muscles, and may even lose the power of standing. The attack may last for two or three months, then pass off, and recur again in the following winter. Progressive wasting of bones and muscles may be present, and rigidity of the knee and ankle joints may supervene, the knee being flexed and the foot pointed. These conditions, although dependent on scurvy, may lead to a diagnosis of rheumatism. In most cases, however, there will be present some other characteristic signs of scurvy, such as sponginess of the gums, hæmorrhages under the skin or elsewhere, œdema of the extremities, and cachexia. Inquiry into the diet will probably elicit the fact that the patient has been having little or no vegetable food. In these chronic or recurrent attacks of scurvy the therapeutic test does not act so promptly as in the more acute cases.

The differences between acute articular rheu-

matism and scurvy can, as a rule, be clearly recognised if a careful local examination is made. In acute articular rheumatism the joints are primarily involved, the swelling is red and inflammatory in appearance, the local temperature is raised, increase of the swelling is marked by further distension of the joint, the effusion is superficial in parts, and fluctuation can be made out. In scurvy the swelling commences near but never in a joint, and presents no signs of acute inflammation; increase of the swelling is marked by extension along the diaphysis of the bone away from the joint, the effusion is deeply seated, and fluctuation can rarely be detected. In addition to the local conditions there will almost always be present some other signs of scurvy or rheumatism which will settle the question. It should also be borne in mind that acute rheumatism rarely if ever occurs during the first eighteen months of life, and that scurvy arises most frequently during this period.

Peliosis rheumatica is a rare form of rheumatic manifestation, which resembles scurvy still further. The purpuric eruptions which characterise it are indistinguishable from those of scurvy, but the other signs—namely, swelling of the joints, endocarditis, &c.—are not found in scurvy.

5. *Other varieties of the hæmorrhagic diathesis*.—In most of these there are some leading signs or symptoms which enable us to arrive at a correct diagnosis.

(1) *Leukæmia*. Microscopic examination of the blood will reveal an excess of leucocytes, which is never found in scurvy.

(2) *Lymphadenoma*. The general and progressive enlargement of lymphatic glands and lymphoid tissue present in this disease does not occur in scurvy.

(3) *Splenic anæmia*. Hypertrophy of the spleen is not present in scurvy. In chronic splenic enlargement, while subcutaneous hæmorrhages and œdema of the eyelids and extremities may occur, there is not subperiosteal hæmorrhage or sponginess of the gums. The therapeutic test will quickly settle the question in doubtful cases, for if the disease is primarily splenic no rapid effect can be produced by an antiscorbutic diet as in scurvy.

(4) *Hæmophilia*. In this affection an hereditary tendency is present, hæmorrhages occur while the child is otherwise in good health, and a common sign is extravasation of blood into the knee or

other joints. These points and the absence of special scorbutic signs will probably suffice to clear up the diagnosis.

(5) *Anæmia*. In the symptomatic form of anæmia a careful examination will usually lead to the discovery of some general cause (tuberculosis, syphilis, rheumatism, &c.), or some local affection (empyema, pleurisy, Bright's disease, &c.), sufficient to account for all the symptoms. In pernicious anæmia, the irregular pyrexial attacks, the diminution in the number of red blood-corpuscles, the absolute pallor of the complexion as contrasted with the dirty yellow colour present in scurvy, and the absence of sponginess of the gums or subperiosteal hæmorrhage, will mark out the disease.

6. *Sarcoma*.—Howard Marsh, Pickering Pick, Northrup and Crandall, and others have met with cases in which scorbutic swellings had been diagnosed as sarcomatous growths. The regions affected were in different cases the gums, the femora, and the orbit. The occurrence of extensive vascular swelling of the gum leading to the projection of dark red masses from the mouth may certainly suggest sarcoma at first sight; but if the case is really scorbutic, the history of the illness and the diet, along with the other signs of scurvy, which if looked for will certainly be found, will usually suffice to make the diagnosis clear. In the case of disseminated sarcomatous growths accompanied by intense anæmia and hæmorrhages in various parts of the body, the diagnosis may be more difficult, as pointed out by Dr. Railton. He relates the case of an infant aged twelve months suffering from this affection, in whom numerous hæmorrhages occurred affecting the skin of the forehead and the eyelids, the periosteum of the femora, and the subcutaneous tissues. This disease is extremely rare, and some of the new growths are usually sufficiently accessible to examination as to allow of a diagnosis being made.

7. *Infantile paralysis*.—A diagnosis of infantile paralysis has been made not infrequently in cases of scurvy. This error arises from the paralytic condition in which the limb or limbs appear to be. In scurvy, as already stated, there is no true paralysis, but a state of *pseudo*-paralysis due to the pain and weakness produced by hæmorrhagic extravasation. Aid in the diagnosis is afforded by the presence of a tender swelling along the bone, of excessive pain on movement of the limb, of

unaltered knee-jerks, and of other signs of scurvy, along with the absence of any evidence of a nervous lesion.

8. *Other affections*.—Certain other affections have been mistaken for scurvy owing to the prominence of some one sign. A diagnosis of spinal disease has been made when the condition was really dependent on rachitic softening and scorbutic hæmorrhage. The appearance of blood in the urine has led to a diagnosis of Bright's disease, swelling and extravasation around the upper end of the femur to one of hip-joint disease, swelling of the gums to one of ulcerative stomatitis, the presence of crepitus and unnatural mobility of bone to one of simple fracture, but a careful examination will always serve to prevent such mistakes.

The Prognosis.

The prognosis in scurvy is extremely good provided that the disease is not too advanced, the hæmorrhages are not very extensive, no special complications exist, and proper treatment is employed. Under these circumstances the results of treatment are amongst the most rapid and striking to be met with in the field of therapeutics. Some authorities hold that the natural course of the disease is towards recovery, and point to the beneficial results which follow from the advent of spring sunshine and warmth, without the adoption of any special antiscorbutic treatment. While it may be admitted that certain patients show a tendency to recovery apart from any direct treatment, even they suffer from a recurrence of the disease if they continue to live under the same conditions, and each attack leaves them more impaired in health. In those cases the usual duration of an attack, when uninfluenced by treatment, is from two to four months (Barlow). The question naturally arises in connection with these cases as to whether the deprivation of fresh (antiscorbutic) food had been partial or complete, intermittent or constant. If it had been only partial or intermittent, one can easily understand how the form of scurvy induced might be chronic or relapsing, and might never present the acute and grave symptoms of the common variety. In my own experience the natural course of the disease, when there had been complete deprivation of fresh food, has appeared to be towards death, either from the

progress of the affection or from some complication.

In the case of an infant the occurrence of extensive hæmorrhage must always be viewed as a serious condition, and when this follows on a period of impaired nutrition and intestinal disturbance, the rallying powers of the patient are often extremely small. An extensive hæmorrhage may lead quickly to a fatal termination, or in other cases the child may die more slowly after a period characterised by a falling temperature, great prostration, general œdema, and increasing cardiac debility. Sometimes cardiac syncope is the cause of death at a comparatively early stage of the affection. Nor is the subsidence of all scorbutic signs to be regarded as an indication that the child is out of danger, for in severe cases the convalescence may be extremely slow, and in others death sometimes ensues, it may be months after the disease was apparently checked.

The Treatment.

The treatment of scurvy is extremely simple, and may be summed up briefly as follows: the employment of a fresh food diet, suitable in quantity and quality to the age of the child. This is to be expected from the ætiological considerations which have already been submitted, and the results of treatment absolutely confirm these statements. Even those who deny that the disease under discussion is really scurvy, admit the remarkable curative effects of dietetic treatment. In describing the details, reference will first be made to the means of preventing the onset of scurvy, and then to the methods of curing the disease when it has appeared.

The preventive treatment of scurvy consists in the employment of a proper diet. In the case of infants, breast milk is the best and the safest diet. There is no case recorded, so far as I know, of scurvy arising in a breast-fed child during the normal period of lactation. The exact changes which occur in the milk after that period are not known, but there are sufficient evidences both in the mother and child that other food supplies are required. If the breast milk fail before this period, then cow's milk, pure and fresh, is a complete diet up to the age of nine months, and, as has been shown, this may safely be modified by boiling or scalding, or Pasteurising. If the infant

does not tolerate milk well, and undigested particles are passed in the motions, it may be advisable to use for a time peptonised, or pancreatised, or sterilised milk, *but this must always be regarded as a temporary proceeding for special reasons.* The addition of plain water or of barley water is, of course, necessary during the early months, but over-dilution of the milk must be carefully guarded against, as this has frequently led to the onset of scurvy. The addition of solid food must not be commenced too early or in too great a quantity at first. A common history is that the child did not appear to be satisfied, and so instead of increasing the strength or amount of the milk, the mother or nurse added some other food, usually of a farinaceous type. By degrees the diet was altered to a minimum of milk and a maximum of starchy food, and thus the way was paved to rickets and scurvy. In all rachitic cases due to faulty diet, it will be found useful to administer a teaspoonful of the juice of oranges, grapes, or lemons twice a day as a means of preventing the onset of scurvy. After the age of nine months a more varied diet is to be allowed; and while fresh milk should still be reckoned an important part of the food for some time, there may be added vegetable soups, potatoes, and fruits, which will serve to prevent the occurrence of scurvy.

The direct connection between the employment of a diet consisting of condensed milk and other patent "infants' foods," and the subsequent appearance of scurvy, has led all writers on the subject to regard such a diet as the chief cause of scurvy. What place are these foods to occupy in the dietary of infants? At the present time these proprietary foods are perhaps more abundant, more advertised, more sought after by parents, and more frequently prescribed by physicians than at any previous period. The question is a large one, but it is daily becoming clearer that these artificially prepared and preserved foods are not to be depended on in the feeding of infants, and in the preventive treatment of scurvy they must be abolished entirely from the diet, as it is practically impossible to confine their use within safe limits, or, in fact, to define what, if any, these limits are.

The curative treatment proceeds on the same lines as the preventive. In the case of infants the proper dilution of fresh cow's milk must be attended to, and all condensed milks or other

"infants' foods" are to be eliminated from the diet. I do not believe that much reliance is to be placed on meat juice or beef tea as antiscorbutics, but these are useful in the treatment of scurvy when digestive troubles are present and milk is not well tolerated. In such cases the use of fresh meat juice for a few days will often rapidly restore the functions of the digestive organs, when a return to a milk diet can be made. At the same time it must be mentioned that Dr. Cheadle, Dr. Barlow, and many other authorities place great confidence in the value of fresh meat juice as an antiscorbutic. In the case of older children a more liberal diet is allowed, attention being paid to the condition of the digestive system, but no salted or preserved foods of any kind are to be given.

The more special part of the treatment in all cases consists in the free administration of fresh fruits and vegetables. The full list of these is a large one, but the following will be found the most useful, namely, oranges, lemons, grapes, potatoes, and cabbages. The juice may be expressed and added to the milk, in the case of infants, half to one ounce daily. Potatoes and other vegetables may conveniently be administered in soup made with meat stock. The voracious manner in which these scorbutic patients devour fruit and vegetables has frequently been noted. In a short time the dull, apathetic infant on catching sight of an orange or some grapes screams with delight and eagerness, clutches at the fruit, and buries its face in the orange or consumes the grapes as rapidly as possible. This indicates, as the result of treatment later on demonstrates, that these infants in the midst of plenty were slowly dying of starvation, the essential element in their diet being rigidly withheld. Care must be taken that the amount of fruit or vegetables is not in excess of the infant's digestive powers, in which case sickness, flatulence, diarrhoea, &c., may follow and interfere with the recovery. In certain cases, more especially amongst older children, there exists a dislike to fruit and vegetables, but this can usually be overcome by a little firm treatment.

The effect of this special treatment is usually rapid and striking. Improvement is often noted within a couple of days, and recovery as regards all acute manifestations within a week, provided

that the disease has not been of long duration. The child begins to brighten up, milk is taken with avidity and relish, the hæmorrhages cease, the swelling of the gums subsides rapidly, the tenderness in the limbs becomes less marked, and soon voluntary movements are performed. By the occurrence of such changes we are able to distinguish between scorbutic and rachitic symptoms, for while the former are rapidly cured or improved under antiscorbutic diet, the latter will persist unchanged for a much longer period. The time for a complete cure in scurvy will depend on the duration of the disease, the amount of hæmorrhage, the general health of the patient, and the presence or absence of complications. If there has been a prolonged period of gastric and intestinal disturbance before the onset of scurvy, the continuance of this condition may delay recovery, and it is not until the patient can take and digest the food that symptoms of improvement appear. I have laid stress on the fact that the diet must be good and nourishing, and must contain a full amount of fresh vegetable material, because some have been content to rely on a small quantity of lime juice as a complete treatment, and have been disappointed with the result.

As regards medicines, most of them are of no special value in the treatment of acute manifestations, and some are distinctly injurious. Owing to a mistaken diagnosis of syphilis, mercury has frequently been employed, and has undoubtedly done harm. If the case be at all doubtful, and if mercury is tried, it ought to be used as a means of diagnosis, and the treatment should only be so far prolonged as to give time to test whether improvement occurs under its use. The presence of complications, such as gastric catarrh, bronchitis, &c., will of course require suitable medicinal treatment. Great prostration must be combated by the administration of stimulants—ammonia, strychnia, and brandy. If the pain is severe, small doses of opium must be given to secure rest and sleep; if sleeplessness alone is present, then chloral, which is well tolerated by infants, is to be preferred. No medicine has yet been discovered which will influence favourably the course of the disease, and in the majority of cases no medicine is required if the proper dietetic treatment is carried out.

As regards local treatment, absolute rest in bed is necessary, owing to the extreme pain and tender-

ness, and the risk of syncopal attacks. The patient will usually give much assistance in carrying out this procedure, so as to avoid the pain which accompanies any movement. In cases with extensive subperiosteal hæmorrhages the patient is to be disturbed as little as possible, and the limbs are to be supported by means of sand-bags. The liability to fracture of the long bones in such cases is very great, their occurrence having been noted from simply handling the limb. Such fractures are to be treated in accordance with surgical principles as far as possible, but the extreme tenderness will prevent the application of splints, and during the acute stage the most that can be done is to keep the limb straight. These fractures show little tendency to unite until the scorbutic condition has been improved, after which healing usually proceeds rapidly.

The general hygienic treatment must be attended to. Sunlight and fresh air are of great value. In the convalescent stage cod-liver oil and iron are useful in restoring the general health. Dr. John Thomson has noted that convalescence may be delayed if the patient is removed from a mild to a colder temperature; in the case of his patient the change having been from an inland town to the sea-side. Active exercise must not be allowed too soon, as fatal cardiac syncope sometimes occurs, and therefore rest in bed ought to be maintained until the patient has been free from marked anæmia and all acute symptoms, and has been under antiscorbutic diet for some time.

Concluded.

CLINICAL LECTURES ON URINE.

Delivered at University College Hospital by

J. ROSE BRADFORD, M.D., F.R.S., F.R.C.P.,

Physician to University College Hospital.

VII.

LAST time we saw that the sugars found in the urine are usually lactose, dextrose, and inosite, but the last is of no great clinical importance. I pointed out also the main points of so-called physiological glycosuria, and the fundamental points as regards the pathology of glycosuria, *i. e.* that it might be due to diabetes and also

to various other conditions, and finally that in diabetes one recognised three fundamental classes of urine. In the first the quantity of urine was increased, with sometimes as much as 10 per cent. of sugar present, a not uncommon one; the second, where the quantity of urine was not much increased, the only departure from the normal, in fact, being the presence of the sugar; and the third where the urine was increased in quantity, and containing sugar but of a low specific gravity. Occasionally in the second class of diabetic urines you have a large quantity of uric acid in the urine, and to this variety the name of gouty diabetes is sometimes given.

In diabetes, I think one may say, if the disease is at all severe, that sugar is not the only abnormal substance present; you get a variety of other bodies, and more particularly compounds of diacetic acid, and very often oxybutyric acid, and occasionally acetone. The presence of acetone in the urine is not characteristic of diabetes; it is often absent in diabetes, and it is often present in non-diabetic patients. There are a large number of diseases in which you have traces of acetone in the urine. In many grave and serious diseases accompanied by marked wasting, and in febrile diseases and after the administration of anæsthetics, a variable quantity of acetone is often found in the urine. Although the name of acetonæmia is given to the toxic condition that supervenes in diabetes and causes coma, yet in this so-called condition of acetonæmia, acetone is not always present in the urine. Just as in the allied condition of uræmia the phenomena are not due to the presence of excessive quantities of urea in the blood, so in acetonæmia the coma is not due to acetone. Acetone is, however, often present in diabetic urine, and it is to this and to the salts of diacetic acid that the urine of diabetes owes its smell. These and other bodies are often present in diabetic urine, and there are certain rough tests for them which we will consider directly. I think I mentioned last time that in the severe forms of diabetes you may get even up to two pounds of sugar excreted in the twenty-four hours; under these circumstances the urine is greatly increased in quantity, and the specific gravity is high, and generally it is over 1035. I want to warn you at the outset that you must not take the specific gravity of the urine as an index of the percentage or gross amount of sugar present. If you have two

specimens of diabetic urine, one with a specific gravity of 1035 and the other with one of 1040, it does not necessarily follow that the percentage amount of sugar in the latter case is greater than in the former; the total amount of sugar passed is of course dependent very greatly on the quantity of the urine passed. The specific gravity is not an accurate index to the percentage amount of sugar present. The specific gravity is in part dependent on other substances present, more particularly on the urea and to a certain extent on the salts and so forth; hence with a urine containing large and variable quantities of urea, the specific gravity will be profoundly affected by it. On the other hand, with a diabetic urine, which is extremely copious, the percentage of urea and salts will be so small that the specific gravity will depend largely on the sugar, and in that case the specific gravity will give you an index to the amount of sugar present. Hence in a case of diabetes if the specific gravity diminishes as a result of treatment, that may or may not be an indication that the percentage of sugar present is less. The three main points about the specific gravity of the urine in diabetes are, firstly, that it is usually high in diabetes; secondly, that the variations in the amount of specific gravity do not necessarily vary *pari passu* with the amounts of sugar present; and lastly, that sugar may be present in a urine with a specific gravity of 1010.

Diabetic urine not uncommonly contains albumen, more particularly in the last stages of diabetes; one of the complications of diabetes is renal disease, and under these circumstances you have albumen in the urine, and with albumen in the urine, particularly if the amount is large, you cannot get the ordinary sugar test, and hence you may think that the sugar disappears when the albumen appears. It is really only that the presence of the albumen for some reason interferes with the ordinary sugar test; hence the rule to look for proteid before testing for sugar.

Another thing as regards the sugar of diabetic urine is, that towards the end of diabetes, particularly if the disease is fatal from coma, the sugar diminishes or even disappears, and you may have a patient dying from diabetes and perhaps for several days or even a week his urine will contain no sugar, and there may be a great diminution in the quantity of the urine. Hence the presence of

sugar in the urine does not prove that the patient has the disease diabetes, since, as we have seen, glycosuria may arise from other causes, and the absence of sugar does not prove that he has not diabetes, since it may disappear during the course of this disease. In the natural course of the disease it is only at the end of the malady that the sugar may disappear from the urine. Of course you will understand that I am only talking of the natural progress of the disease, and I am not alluding to the effects of treatment. I have known of one diabetic patient where the sugar disappeared temporarily from the urine without treatment and without coma supervening; but it is usually a very fatal sign, and the sudden diminution or disappearance of the sugar in a diabetic patient may mean that coma is imminent, and coma in diabetes is practically always fatal.

Patients with diabetes tend to have a subnormal temperature, just as is the case with patients suffering from renal disease. If a patient with diabetes contracts some febrile complaint, one of three things may happen: firstly, the onset of the febrile illness may cause the development of coma; secondly, the febrile malady may run the whole of its course without any fever; and, thirdly, during the course of the febrile complaint the sugar may entirely disappear from the urine. I cannot do better than to call to your mind a case of Dr. Poore's. A man with severe diabetes contracted typhoid fever, and was ill for two or three months, and during the whole time he was feverish there was no sugar in his urine. This is a point of some theoretical importance; the fundamental thing in many cases of diabetes is that the excess of sugar in the blood may be due not so much to increased production as to the sugar formed in normal amount not being used up, and many persons have supposed that in ordinary diabetes the patient cannot use up the sugar that he normally makes. The fact that the glycosuria disappears during pyrexia seems to show that this sugar can thus be used during the febrile process.

There are various grades of severity of diabetes, more particularly the following:—Patients whose sugar disappears as soon as they are dieted, the sugar disappearing as soon as the carbohydrates are withdrawn; and then you have patients in whom the withdrawal of the carbohydrates has little effect, and on whom treatment by opium has

effect; and finally there is a severe form in which opium and diet have little or no influence, and these patients often pass very large quantities of sugar, and frequently are under twenty years of age.

As regards the other bodies present in the urine in diabetes, acetone, diacetic acid, and oxybutyric acid are the more important. Some say that they are only present when coma is present or imminent; but that, however, is not the case, and diacetic acid and even acetone are often present in diabetic urine without coma. For diacetic acid we have a rough and ready test with perchloride of iron, by which its presence can generally be detected. The recognition of oxybutyric acid is more difficult, because there is no rough test for that; you have to distil the urine and obtain the oxybutyric acid, and determine its melting point, and so on. There is no doubt that oxybutyric acid is present when coma is present or imminent, but I do not think that one can say that it is not present in other conditions in diabetes when coma is not necessarily imminent.

Now as regards the tests. You have of course the qualitative and quantitative tests, and there are a large number of qualitative tests, but there are not many with which we need concern ourselves; first of all, there is the well-known copper test. I only repeat that more mistakes are made in testing for sugar than in any other form of test. The best way is to pour sulphate of copper into a test-tube, and then empty this sulphate of copper out; you will have enough of the salt left sticking to the tube, then put in the urine, and then an excess of potash, and then boil it. There are two precautions; one is not to have too much copper present, and the other is, to use the expression of a former professor of chemistry, "do not cook it," raise it simply to the boiling point. If the urine is boiled a long time, almost any urine will give a slight reaction; simply raise the fluid to the boiling point. Supposing the urine contains but little sugar, if you boil the urine with an excess of copper sulphate you may get some black oxide of copper formed, and under these conditions you cannot tell if sugar is present or not. The object of only raising it to the boiling point is that uric acid, urates, and particularly creatinin, have a slight reducing action on copper, but which is not marked unless the urine be boiled for a long

time. If you take any dense febrile urine and boil it with copper and potash you will get a spurious reduction. Fehling's solution is a more convenient method for performing the same test; in this reagent, the hydrate of copper is kept in solution by the tartrates in the Fehling's solution. The objection is that Fehling's solution does not keep, and you ought to take the precaution of always boiling it by itself first of all to see that it does not reduce. Take a big test-tube and boil thoroughly the Fehling solution, it does not matter how long you boil that; you add to that preferably one or two drops of the urine, do not add too much because an excess cools down the boiling mixture. If there is an appreciable amount of sugar present, the reduction occurs at once; if there is only a small quantity of sugar present, you do not get the reduction for a few moments; if there are only traces, it may be necessary to add more than the few drops of urine, but under no circumstances add more than half a volume. If you add these larger quantities of urine you have to raise the fluid to the boiling point again.

There is the well-known potash test, the so-called Moore's test, in which on boiling the urine with potash the liquid becomes brownish-red in colour, varying from a pale sherry to a dark mahogany colour, depending on the amount of sugar present. It is a very good test, but not so delicate as the copper test.

There are a large number of other tests, for instance the picric acid test. I think this test has this advantage—it is a test for albumen and for sugar, and the presence of the albumen does not interfere with the sugar test. You take your suspected urine, and add some saturated solution of picric acid, and add some potash, and you warm it; if sugar is present you get a very deep blood-red colour, but the fallacy of the test is that every urine gives a red colour with picric acid and potash; and it is simply a question of the depth of the colour. It is not a question of the colour alone, for creatinin gives the colour; therefore you must do a control test with a urine that does not contain sugar. In the hands of experts it is a very good test, but for everyday purposes it is not to be recommended, for if you rely on it exclusively you will no doubt one day find sugar where no sugar is present. The phenyl-hydrazin test is far less used now than it used to be; there are two reasons for

that: in order to do it properly you have to do it with a water bath, and it takes time, and when you do it properly it is, if anything, too delicate. It is a test which is useful in testing for traces of sugar in normal urines. Hence the phenyl-hydrazin test, though a very excellent test, is not a test that is really of any very great practical importance.

There is another very good test for practical purposes. It is a bad test for determining the amount of sugar, but it is a very good test for determining that sugar is present—I mean the fermentation test. The copper test comes off with a number of bodies which are not sugar, whereas the fermentation test does not. You simply take a test-tube, fill it absolutely full with the urine, put a little piece of yeast into it, turn it upside down, preferably over a vessel containing a little mercury, or over a little vessel containing the same urine, and then you put by the side of it a test-tube similarly arranged, and containing some urine without any yeast; put it in a warm place, preferably a warm chamber, and gas accumulates at the top of the test tube. It is a useful test in cases of doubtful glycosuria; a patient comes to you with vague symptoms, and on boiling the urine you get a sort of a reduction, say a greenish-red colour, but you do not feel sure if it is or if it is not sugar, and you think perhaps that it may be uric acid, or creatinin, or what not, and under these circumstances the fermentation test is useful to satisfy your mind as to the presence or absence of sugar, but it is not so useful to determine the amount of sugar. It can be used for that purpose; you can put some yeast into the urine, and take the specific gravity before and after, and then there are tables which will give you the amount of sugar for each unit of specific gravity lost. There are plenty of circumstances under which you may be able to ferment the urine when you may not perhaps be able to procure Fehling's solution.

As regards the fallacies, there are two sets; you may find sugar when it is not present, and you may fail to find sugar when it is present. The first is the more common. First of all there are drugs, more particularly the salicylates, which are excreted in the urine in the form of salicyluric acid, and that body reduces copper; salicylates and salicin are taken largely nowadays. That is the drug on the whole that you are most likely to come to

grief over. Glycuronic acid, a body normally present in the urine in combination with various aromatic bodies, reduces copper. There are a large number of aromatic bodies excreted in the urine; they are in combination with sulphuric acid or with glycuronic acid, and under certain circumstances, more particularly after the administration of camphor and chloral, the amount of glycuronic acid is greatly increased, and glycuronic acid gives a very perfect sugar reaction, and you can only diagnose between this and dextrose by the fermentation or by the phenyl-hydrazin test. Urates and creatinin in highly concentrated febrile urines may prove a fallacy by your having too much urine in the tube, and heating it too long. If you follow the rule of not giving a definite opinion until you have fermented the urine, you will not go wrong in these cases. As regards not finding sugar when it is present in the urine, the principal error is the presence of albumen interfering with the sugar test, and it is an error which may be avoided by testing for albumen first.

As regards the estimation I am not going to describe fully the process, I only want to mention some of the pitfalls. The estimation of the quantity of sugar is not an easy thing to do correctly, simply largely owing to the fact that men are not told the possible fallacies. In doing a quantitative estimation, supposing you use Fehling, the following are the main points:—You should take your quantity of Fehling's solution, and that you should dilute very freely; take your 10 c.c., dilute it freely, —usually it is diluted to 50 c.c.; you must freely dilute your urine also, because the amount of sugar may be very great in the urine, and a drop from the burette may be too much at a time. You cannot, as a rule, determine the amount of sugar correctly by doing a single estimation. If you are given a urine to examine, you must make at least two determinations, and you must therefore boil up your Fehling and run in your diluted urine rapidly; the essence of success is to do the thing quickly, you run it in rapidly; if you have no idea as to what the amount of sugar in the urine is you must run it in in quantities of 2 or 3 c.c. If you have run in say 30 c.c. you take a fresh lot of Fehling and start again; you run in 20 c.c. at once, and then only small quantities, and then perhaps you may hit it off to within a c.c. But it is absolutely impossible to do a sugar estimation

with a single observation. If you try to do it as most men do, this is what happens—the urine is run in in small quantities; a considerable amount of time is taken up in the process, and the blue colour returns to the liquid on cooling. Further quantities of the urine are run in, and so on, and ultimately an extraordinary result is obtained. It is not so simple to determine when the blue colour goes. It is often really very difficult. If you want to determine it, you must briskly boil when the urine is run in. Take the lamp away, the precipitate subsides, and then tilt the dish; if it is only carelessly warmed you have to wait for the precipitate to subside, and you get the most extraordinary results. If you allow the liquid to cool the blue colour comes back, and so it goes on, so that the essence of success is to have the Fehling briskly boiling, to run the urine in very quickly, not to delay matters, and then to observe the presence or absence of the blue colour almost at once. When Mr. Gerrard was the dispenser at this hospital he introduced a modification of Fehling's solution, containing cyanide of potassium; it was really a very good method indeed, as far as I know. Owing to the presence of the cyanide no red precipitate was formed, and the end of the reaction was shown by the disappearance of the blue colour from the liquid. The difficulty arises from the conflict of the red precipitate and the blue colour.

You can form no opinion on a diabetic case as regards either its severity or the effect of treatment unless you make a routine of doing a quantitative estimation of the sugar daily. If you are going to quantitatively estimate the sugar you must have a twenty-four hours' urine—that is a *sine quâ non*. As regards lactose, this sugar is present; in the urine of suckling women it is only of theoretical importance, and it does not ferment with yeast, so you can detect its presence by the phenyl-hydrazin test. As regards the other bodies in diabetes, acetone and diacetic acid and oxybutyric acid, we will take them in that order. Diacetic acid is recognised by the red colour it yields with ferric chloride. Take one drop of the ferric chloride and add it to a large quantity of the urine; a precipitate of ferric phosphate falls, and the urine assumes a reddish or mahogany colour: that is not due to acetone, it is due to diacetic acid. The best way to detect acetone in the urine is to

make iodoform, and the simplest way of making iodoform in order to determine the presence of acetone is to use a drachm of liquor potassæ, and dissolve in it twenty grains of iodide of potassium; boil it in a test tube thoroughly, float on to the top of it from a pipette about an equal quantity of the urine. If any appreciable quantity of acetone is present you have yellow crystals at the junction of the two liquids, viz. iodoform. The ferric chloride test comes off in a very large number of diabetic urines, but the acetone reaction does not come off in such a large number. Oxybutyric acid is of no great clinical importance, because there is no ready method of detecting it. You must pursue a more or less elaborate distillation of the urine and collect the distillate, and separate the acid and observe its melting point. The main interest of this body is that a considerable number of observers consider that coma is due to the oxybutyric acid. It has long been known that the urine and even the blood of diabetic patients may be highly acid, so that the main importance of it is rather from that point of view than from a clinical point of view, inasmuch as there is no rough test to detect its presence.

THERAPY AND DOSAGE OF MARMOREK'S SERUM.

DR. COX, in the 'Journ. of the Amer. Med. Assoc.,' September 11th, 1897, says that by far the most important division of this subject is the one which treats of clinical data. In this we can speak with greater confidence, notwithstanding the fact that we are occasionally confronted by theoretic clashings which tend to make our position at first glance appear somewhat paradoxical. For example, an ordinary acute abscess seems to bear very little resemblance to a case of erysipelas, especially a case of erysipelas in which suppuration does not occur; and yet it is admitted on all hands that the pathogenic germs of these two affections are identical, one being the "*Streptococcus erysipelatis*" of Fehleisen (1883), and the other "*Streptococcus pyogenes*" described by Rosenbach (1884) and Passet (1885). Again, puerperal septicæmia caused by streptococcal infection and chronic inflammation of the middle ear, do not seem to stand in very close relation to each other, and neither of them

would ordinarily be placed in the same class with either of the two diseases just mentioned; and yet we have in all of them the same pathogenic force at work, and all of them readily yield to the same line of treatment.

It would be impossible to enumerate all the diseases in which the streptococcus figures, either as the principal or as a complication, for the simple reason that in all probability not a tenth of them are known; but it is sufficient for us to know that wherever and whenever found it is a danger signal of weighty import, and that immediate steps should be taken for its destruction. It is no argument to say that because certain so-called "kinds" or "species" of the germ are harmless we would ever be justified in waiting for a demonstration of their virulence. If the deductions of Widal, Marmorek, and others who believe in the unity of the germ are correct, the most benign of them may, by change of conditions, soon become the most virulent.

In order that I may not be misquoted or misunderstood, I wish to distinctly state that the antistreptococcus serum is only recommended for streptococcic infection, and not for diseases or conditions in which this infection does not exist. Some cases of puerperal septicæmia are caused by the *Bacillus coli-communis*, and in the treatment of these the serum would be without effect. But in the greater number of cases chains of streptococci are found in large numbers, and these yield to the serum treatment in a most remarkable manner. Hence we emphasise the necessity of a bacteriologic examination in every suspected or doubtful case, in order that treatment may be pursued upon rational and scientific lines. As a general rule there is ample time for thus settling the question of diagnosis before treatment is begun; but in occasional instances where the gravity of the case would render delay dangerous we would advise yielding to empiricism to the extent of administering at least one efficient dose of the serum without waiting for the bacteriologic test, knowing that in any event no harm would follow, while, as has frequently been the case, it might be the means of saving a human life.

When we say that erysipelas may be aborted by the use of this remedy, we speak from authority furnished by the case-books of reputable physicians. To say that it can always be aborted would probably

be a flagrant error; but that it may always be modified, ameliorated, and abridged is the almost universal verdict of the profession of two hemispheres. If its value as a therapeutic agent had to be measured by its power over one such disease as puerperal septicæmia, it would doubtless retain its place in the list of valuable remedies for all time to come. But such is not the case. We already have such an overwhelming array of proof of its efficacy in a number of apparently diverse conditions as to lead to the belief that it will soon be esteemed as the most valuable of the blood serums yet discovered. Besides the diseases already mentioned, in which it may be relied on to effect a cure with little or no other treatment, it has been used with remarkable success in many cases of mixed infection, removing with great promptness an ugly complication and rendering a disease amenable to ordinary treatment, which would otherwise have resulted fatally. Among such diseases may be mentioned diphtheria, scarlet fever, bronchitis, broncho-pneumonia, and phthisis pulmonalis. In the last-named it has been especially useful, as will appear in the report of cases to follow.

It is claimed by several observers that, no matter what the disease may be, the use of the serum is always followed by increased leucocytosis. If this be true, and the army of fighting phagocytes can be recruited at will, what mind can foretell the future, even the near future, of this infant that we designate serotherapy? It is already one of the recognised branches of legitimate medicine in the Old World, and is rapidly gaining in popularity here. The diphtheria antitoxin is no longer an experiment with us, and considering that the Marmorek serum was only introduced into the United States less than a year ago, its success in this country must be regarded as little less than phenomenal.

In France, where the serum has been used most extensively, the weight of evidence is vastly in its favour. True, it has been severely criticised in a few instances, but a careful study of such cases has generally resulted in victory, rather than defeat, for the serum. They have almost invariably been cases of mixed infection in which the serum was expected to exhibit powers not attributed to it by its discoverer, and, very generally too, when it was given in *inadequate doses*. In some instances, too, where adverse opinions were expressed, an ad-

mittedly weak or doubtfully prepared serum was used. Thus in the 'Botkins (Russia) Clinical Gazette,' No. 43, 1896, we find a report of sixteen cases of scarlet fever treated by Dr. L. Rappaport in all of which a serum was used. This series of cases he divides into three groups, as follows: four of "medium gravity," two "severe," and ten cases "with various complications." The serum employed was of Russian manufacture, and admitted to be much weaker than Marmorek's; and as 20 c.c. was the maximum dose given in any one case, it seems but natural that some of the cases should die "without," to quote Dr. Rappaport, "the serum having had any apparent effect upon their course." Very different from this report is the one of Dr. Rondot, made at the meeting of the French Medical Congress held at Nancy, France, August 6th, 1896. In this report Dr. Rondot gives his experience with Marmorek's serum in the treatment of a large number of cases of erysipelas. He invariably found that the serum produced a rapid improvement in the general condition, and marked reduction of temperature and diminution in the duration and gravity of the disease. In the most severe cases there was always rapid and pronounced retrocession in the acuteness of the lesions.

At the meeting of the Ophthalmologic Society of France, November 3rd, 1896, Dr. Boucheron related his successful use of Marmorek's serum in the treatment of purulent dacryocystitis and other cases of streptococcic infection of the eyes. He, in common with Widmark, Morax, and others, had noticed that many cases of dacryocystitis were caused by the streptococcus, and that they were always of a well-defined type. The most rebellious of these are those that he specially commends for the serum treatment. Boucheron also found that this serum could be successfully used as a preventive of streptococcic infection of the ocular organs at the time of operation for cataract or ocular traumatism.

Drs. Vahle, Steffek, Döderlein and Walther all found the streptococcus present in the vaginal secretions of such a large proportion of pregnant and parturient women that Dr. Vahle arrived at the conclusion that before every accouchement, even in healthy subjects, prophylactic injections of the serum should be made. ('Zeit. für Gebur. und Gynec.,' vol. xxxv, No. 2.)

At a meeting of the Medical Scientific Society of Lyons, France, Dr. Vinay reported thirteen cases of puerperal fever treated with Marmorek's serum, nine of which were eminently successful. This is not a record to be ashamed of; but it is highly probable that with more extended experience in the technique of administering the serum since these cases were reported, the percentage of recoveries would be considerably increased in a similar series at this time.

Dr. Sevestre, in charge of the diphtheria patients in the Sick Children's Hospital, Paris, uses Marmorek's serum freely in all cases of mixed infection, with excellent results, the proportion of recoveries being considerably augmented since the employment of this remedy.

M. Cuffer reports a remarkable recovery after the use of twenty-eight vials of the serum extending over a period of one month. The case was one of endocarditis, pericarditis, congestion of the lungs with pleurisy, arthritis and endometritis. He also reports a case of acute pelvic inflammation in which the left broad ligament was infiltrated, forming a very large tumour. Injections of serum prevented the formation of pus in the tumour, and all unpleasant symptoms rapidly disappeared.

Dr. Chantemesse, of Paris, had remarkable success in the treatment of 411 cases of erysipelas in which he used the serum. In every instance the symptoms were most favorably influenced by the treatment; and after deducting three cases which were absolutely hopeless on account of complications, the recoveries were 99.3 per cent.

Dr. Van Arsdale, of New York, reported two interesting cases, one of appendicitis with general purulent peritonitis, and the other gangrene of the gall-bladder accompanied by sero-purulent peritonitis. Both were extremely grave, but under appropriate surgical measures coupled with the use of the serum, recovered.

Dr. Henry W. Berg, of the Willard Parker Hospital, has reported so many instances of the successful use of the serum in cases of diphtheria and scarlatina where there was mixed infection, that his experience must be familiar to most practitioners, and it is therefore only necessary to mention it here.

The 'Medical Record,' March 14th, 1896, and the 'Medical News,' April 4th, 1896, both give glowing accounts of the efficacy of Marmorek's

serum in the complications and sequelæ of scarlet fever. Adenitis, otitis and albuminuria are among the complications mentioned by these authorities, and all of them were speedily controlled by the serum.

Dr. Howard Lilienthal ('Medical Record,' March 20th, 1897), reports a case of a child supposed to be suffering from rheumatism of the hip-joint. The doctor opened an abscess, found chains of streptococci, and used the serum with success.

Dr. Gerster corroborated Dr. Lilienthal, and said he had used the serum in a number of cases at Mt. Sinai Hospital, including such diseases as peritonitis, appendicitis, and general sepsis, and all with excellent results, some of the recoveries being regarded as marvellous. Dr. Richards in the same journal reports the successful use of the serum in a case of puerperal septicæmia in which the prognosis was very bad.

Reasoning from analogy it may be supposed that the gynæcologist will find in Marmorek's serum a sheet-anchor in his speciality. He finds pus everywhere. It confronts him in the reproductive tract from the ovary to the ostium vaginæ, and is one of his most dreaded enemies in the connective tissue throughout the pelvic cavity. It cannot be possible that all of these are of gonorrhœal origin. Many of them must be sequelæ of simple congestions brought about by innocent and unavoidable causes. If Marmorek's serum controls this condition when applied in the congestive stage in other localities, why not also in this hotbed of abscesses? We believe it will do so if intelligently employed, and we believe also that laparotomy, so frequently resorted to in the past, may often be rendered unnecessary by this simple and easy means.

In conclusion, we will refer to the use of Marmorek's serum in cases of tuberculosis, having purposely reserved mention of this disease till the last on account of its vast importance. A little more than a year ago Dr. Charles T. McClintock, of Ann Arbor, read a paper before the Michigan State Medical Society, in which he predicted that the antistreptococcic serum would find its greatest usefulness in cases of tuberculosis. That was the first published utterance to this effect, and Dr. McClintock has proved himself a prophet. During the month of July, 1896, Dr. W. H. Weaver, of

Chicago, used Marmorek's serum in three such cases, reporting them in the 'Journal of the American Medical Association,' September 5th, 1896. That was the first published report of cases treated in this way. Since that time a number of cases have been so treated by physicians in different parts of the country, but little or nothing has been published concerning them. Why, it would be difficult to say. The importance of the subject would seem to merit more attention than it has received. In all the published reports, however, and in all received from private sources, there has been a pleasing unanimity in the action of the serum. Without exception it has done all that was expected of it. That is to say, it has invariably destroyed the streptococcus microbe, and thus freed the case from a disagreeable and dangerous complication. Having thus converted the case from one of mixed infection into one of simple tuberculosis, the system is left in better condition to resist the ravages of the tubercle bacillus, and consequently more likely to respond to appropriate treatment for the destruction of this germ. The serum should be used in those cases only where the bacteriologic test shows the presence of the streptococcus microbe. In all such cases there is liable to be considerable cough, purulent expectoration, night sweats, high temperature, insomnia and anorexia, all of which are greatly modified, and sometimes disappear altogether, under the influence of the antistreptococcus serum. Oftentimes the train of symptoms just enumerated will be found where there is no streptococcic infection. These, as a rule, are very grave cases, and the serum treatment is not indicated. Lack of space forbids a detailed account of the progress of the different cases in which the serum has been used; but suffice to say that while some of them were hopeless as far as ultimate recovery was concerned, yet without exception there was such decided amelioration of distressing symptoms as to lead to the conclusion that earlier employment of the serum would at least have resulted in greatly prolonging life.

Marmorek's serum should always be used by hypodermic injection into the cellular tissue. If due antiseptic precautions are observed, and ordinary skill used in its administration, it is free from danger, and the dose is practically without limit.

In all grave cases, such as puerperal septicæmia,

and especially if treatment has been considerably delayed, an initial dose of 30 c.c. is advised. This may be followed by doses of 10 c.c. or 20 c.c. every twelve or twenty-four hours, according as the symptoms are affected.

In ordinary cases of erysipelas the initial dose is 20 c.c., and in many instances may be all that is required. However, it may be repeated once or twice in twenty-four hours if necessary. In the complications of diphtheria and scarlet fever one dose of 10 c.c. will be found sufficient in the vast majority of cases. In the mixed infection of tuberculosis, the dose should ordinarily be 10 c.c., to be repeated every second or third day until the microbes have disappeared from the sputum, usually requiring from four to six injections.

In all other affections, such as acute abscesses, pelvic inflammations, chronic inflammation of the middle ear, and in suppurative processes generally where the streptococcus germ is found, the dose must be regulated according to the exigencies of the case.

Children bear the serum remarkably well, and 10 c.c. is the usual dose for children of all ages.

Almost any part of the body where the skin is not tightly drawn may be selected for the injections. In adults preference has usually been given to the lumbar or gluteal regions; while in children the lower abdomen, a little to the right or left of the median line, is more frequently selected. A perfectly sterile syringe and needle should be used; and unless some special condition exists to call for more thorough cleansing, the site may be sufficiently prepared by a careful bathing in alcohol.

Conclusions.—1. In Marmorek's serum we have a remedy of the greatest therapeutic value. 2. So far as known, it is only applicable to streptococcic infection, simple or mixed; hence it naturally follows that—3. An early bacteriologic examination should be made in order to settle the question of diagnosis and point the treatment. 4. Its action upon the microbe is rapid and certain if given in adequate doses.

NOTES.

Disinfection by Formaldehyde Vapour.—If one may trust the reports of careful observers in

Europe and America, and may judge from a limited personal experience unchecked by laboratory investigations, the problem of disinfection of apartments and their contents, during and after their occupancy by patients having contagious diseases, has at length been solved. Dr. Hans Aronson (*'Zeitschrift für Hygiene und Infektions-Krankheiten,'* vol. xxv, June, 1897) summarises investigations to date upon the antiseptic properties of formaldehyde, and recounts new experiments tending to confirm his original researches. Various methods of disinfection by utilising the penetrating powers of formaldehyde gas upon a large scale, have been proposed. Aronson finds most of them objectionable, including in this category all attempts at vaporisation by means of heat from commercial solutions of formaldehyde in water, or from solutions in methylated alcohol, and attempts to develop the gas directly by heat applied to wood alcohol in lamps of various devices.

Of all methods he prefers that of Schering, which utilises the solid polymerised formaldehyde (paraformaldehyde or paraform) in the shape of pastils weighing 1 gramme each. These being exposed to heat in lamps of proper construction, the heated gases of combustion convert the paraformaldehyde again into formaldehyde gas, which becomes thoroughly mixed with them, and with them is distributed into all portions of the place to be disinfected; the necessary moisture being likewise supplied by the combustion. In a room of about 100 cubic metres (3500 cubic feet) content ($25 \times 13 \times 10\frac{1}{2}$ feet) he placed test objects at different levels. The microbes used were staphylococcus, streptococcus, *Bacillus pyocyaneus*, typhoid, diphtheria, and tubercle bacilli, and anthrax spores. It was found that one lamp containing 100 pastils (one pastil per cubic metre—35 cubic feet) was sufficient to destroy all germs but anthrax spores. For this 200 pastils sufficed.

It made no difference where the test objects were placed, or through how many thicknesses of material the vapour had to penetrate. Even the scrapings of dust were sterile. The necessary number of pastils were placed in the apparatus, the lamp lighted, and the room closed for twenty-four hours. A strong odour of formaldehyde perceived on entering was quickly dissipated by opening the windows.

In cases of diphtheria and scarlet fever we have

had the small lamp with a single pastil used continuously, while likewise vapourising a mixture of turpentine and eucalyptol, or the latter alone, for the sake of the odour as well as for the undoubted therapeutic usefulness of the latter mixture.—S. S. C. in the *Philadelphia Polyclinic*, September 11th, 1897.

The Therapeutic Value of Nebulised Fluids.—F. T. Rogers, in the 'Atlantic Medical Monthly' of March 20th, 1897, contributes an article with this title. After describing the nebulisers usually employed he tells us that he can recall numerous cases where the inhalation of the nebulised solution of various antispasmodics promptly relieved asthmatic paroxysms, and with greater ease of application than was possible by the ordinary method of fumigation. Of late years the writer has not seen this disease in his practice, but has had ample opportunity in his own family to test the efficacy of the treatment.

The following solution, nebulised and inhaled, has given the writer the greatest satisfaction :

R Antipyrin	gr. xv.
Pyridine	3j.
Sodium nitrate	3ij.
Tincture belladonna	3v.
Tincture lobelia	3v.
Tincture stramonium	3v.
Tincture ipecac.	3v.
Glycerine, q. s.	...	ad	3iv.

Relief has been instant and prolonged, and in two cases of periodic hyperæsthetic rhinitis with asthma the solution used in one of the hand nebulisers has proved efficient, and has not had the usual loss of efficacy upon repeated trials which so commonly is found with almost any antiasthmatic mixture.

The systematic use of the nebuliser with forced inspiration offers to the asthmatic a double value of exercise and medicine.

Every man, whether engaged in general¹ or special practice, is likely to be asked at some time to quickly relieve the hoarseness resulting from an acute or subacute laryngitis, and more frequently that due to its chronic form. The use of cocaine in spray followed by the topical application of nitrate of silver or chromic acid is the classical treatment, and is the most efficacious in the majority of cases ; but Rogers has also gained excellent

results by the use of a nebulised fluid containing these drugs, and it has the great advantage of producing quite as much effect on the mucous membrane of the larynx, without the oftentimes distressing spasm which results from applying a caustic solution.

In cases of public speakers, singers, or actors it has a further advantage—the possibility of auto-application ; and when tried just before the effort of speaking or singing is much more efficient than is possible for a topical application to be when made at the office several hours previous.

In acute coryza—that is, in certain cases—the effect the writer has gained from the use of a 4 per cent. solution of antipyrin has been marvellous, and the contractile effect upon the engorged mucous membrane is not only quite as persistent as that of cocaine, but has none of the disadvantages of that drug.

Too many of the abortive forms of treatment for colds depend entirely upon cocaine for their efficacy ; and the danger of toxic effects, and even the formation of the drug habit, are powerful reasons why this drug should not be used.—*Therapeutic Gazette*, August, 1897.

Nervous Cough.—Koch reached the following conclusions :—1. The existence of a true nervous cough cannot be denied. 2. This cough, emanating from a nerve centre, may be diagnosed as such when abdominal and thoracic organs are intact, when one can exclude hysteria, whooping-cough, and beginning phthisis. 3. The monotonous, involuntary cough, always the same in each patient, forms the principal symptom in this affliction. 4. Medication fails. The cough ceases spontaneously after a sea voyage or a trip to the mountains.—*Medical Record*, Sept. 18th, 1897.

Night Terrors.—Braun, after critically discussing the existing theories on favor nocturnus in children, declares it to be a disease by itself, which is closely allied to the conception of neurasthenia, *i. e.* "an irritable weakness." Following this, a description of the characteristics of the attack and their demonstration is given. The sudden jumping up of the infant out of its sleep—symptomatic especially in colic—has no relation to night terrors. The etiology as well as the treatment is that of neurasthenia, and the latter should be pointed in the direction of nutrition and education.—*Der Kinderarzt*, 1897.

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CASES ILLUSTRATING THE SURGERY OF ABDOMINAL VISCERA.

BY

J. BLAND SUTTON,

Surgeon to the Chelsea Hospital for Women, and
Assistant Surgeon to the Middlesex Hospital.

DURING the past summer the following cases illustrating the surgery of the spleen, kidney, stomach, pancreas, and liver came under my care, and as they possess some unusual points of practical interest I have ventured to publish them.

Pelvic Cellulitis ; Residual Abscess ; Splenectomy.

In May, 1897, Dr. Chapman Grigg placed under my care Mrs. —, æt. 35 years, on account of a swelling which he regarded as an abscess in the left ilio-costal space. This swelling was as big as a fist, and seemed fixed in the abdominal wall, but it tailed away indefinitely, and simulated very closely a renal tumour. Twelve months previously the patient had been delivered of a child, and this event was followed by an inflammatory effusion into the left mesometrium. The effusion gradually subsided, and in the course of convalescence a swelling appeared in the left lumbar region, accompanied by pain and an average daily temperature of 100° Fahr.

The patient was admitted into the Chelsea Hospital for Women, and arrangements made for incising the swelling. On making an incision over the most prominent part of the swelling I found in the subserous tissue a collection of caseous material, and following this backwards it led me to the colon. On attempting to ascertain its limits my fingers entered some soft tissue which bled very freely. This mass felt so like the kidney that I suspected the whole mass to be a pyonephrosis, and the bleeding continuing I carefully enucleated the lower border of the soft mass and drew it into the wound in order to control the

hæmorrhage. This manœuvre tore the tissue, and to my surprise I found that I had broken off the lower third of the spleen. I at once transfixed the pedicle with silk ligatures and removed the torn fragment. The escape of venous blood from the upper part of the spleen was so free that in order to arrest it I found myself compelled to remove the remainder of the organ.

On returning my attention to the abscess cavity I succeeded in removing much caseous pus, and discovered that the descending colon formed the floor of the abscess, and, to my disappointment, found a hole in the colon capable of admitting an index finger. The gut was too soft to permit of suture, so I united it with thin silk to the peritoneum and freely rubbed iodoform into the walls of the abscess, introduced a large drain into the abscess cavity, and another into that part of the wound communicating with the colon. The incision was then secured with silkworm-gut sutures.

The patient bore the operation extremely well ; fecal matter escaped from the lower wound during three weeks, and she returned to her home with a very narrow sinus discharging a few drops of pus daily. Three months later there was still a narrow sinus maintained by a buried suture.

In this case it would seem that the pelvic cellulitis extended along the mesorectum and upwards to the descending mesocolon. This formed an abscess which was limited by the colon and the spleen.

I venture to put the case on record for two reasons :—1. Because an abscess in such a situation is an unusual sequel of pelvic cellulitis. 2. The remarkable and rapid convalescence the patient made after what was practically a colotomy and a splenectomy.

At this date the health of the patient is excellent, and she has not experienced any inconvenience from being spleenless.

A Renal Tumour probably arising in an Accessory Adrenal.

A widow, 58 years of age, was placed under my care at the Chelsea Hospital for Women, on

account of a large tumour occupying the left side of the belly.

The woman seemed ill, had a cough and a small amount of albumen in the urine, but no blood. The tumour was moveable, solid, and resembled in outline a spleen, but was devoid of notches; it could be pushed into the loin, but yielded a dull note to percussion in front. The blood-corpuscles were normal in amount, and in right proportion.

The diagnosis was doubtful, and as there seemed to be constitutional disturbance as well, Dr.

section measured in its major and minor axes 18 and 14 cm. respectively. When fresh it looked like a large extravasation of blood beneath the renal capsule, but on microscopic examination its tissue resembled the zona fasciculata of the adrenal, and contained many large, double, and multi-nucleated cells, resembling those found in the adrenal of the *foetus*.

Since Grawitz drew attention to tumours of this nature many examples have been recorded, and they nearly all agree in the following points:—

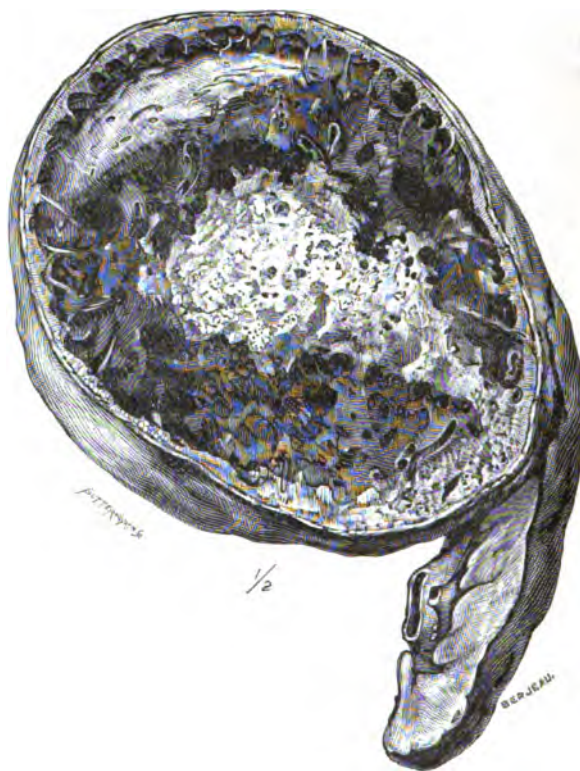


Fig. 1.—Tumour and the kidney in sagittal section.

Coupland kindly took charge of the case at the Middlesex Hospital. Under his care the general conditions greatly improved, but the exact nature of the tumour remained doubtful. The patient was seen by several surgeons, who regarded the "swelling" as an enlarged spleen.

Eventually I performed coeliotomy, and found the tumour to be connected with the upper pole of the kidney. The tumour and kidney were removed, and the patient made an excellent recovery. The relation of the tumour to the kidney is shown in Fig. 1; together they weighed $1\frac{1}{2}$ kilos., and on

they are distinctly encapsuled and occur in adults, and do not give rise to hæmaturia, although the tissue of the tumour is often infiltrated and torn by extravasations of blood. It is also an important fact that the adult adrenals are liable to tumours of similar character, in which proneness to extravasations of blood is a marked feature.

It is well known that accessory adrenals have been observed not only beneath the capsule of the kidney (Fig. 2), but beneath the capsule of the liver and between the layers of its falciform ligaments; also in the course of the spermatic artery.

Marchand has also detected them in the neighbourhood of the ovaries of fœtuses, and his observations have been confirmed (Fig. 3).

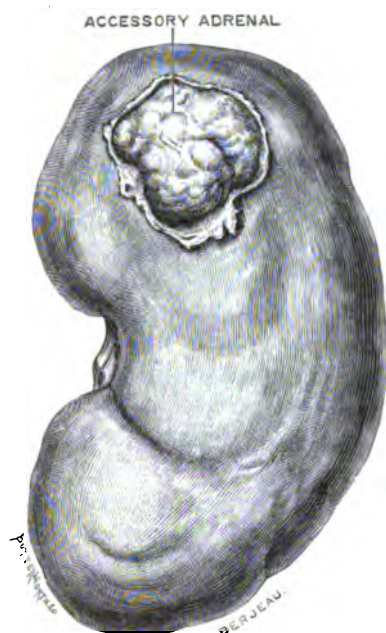


Fig. 2.—An accessory adrenal beneath the capsule of the kidney. (Museum of the Royal College of Surgeons.)

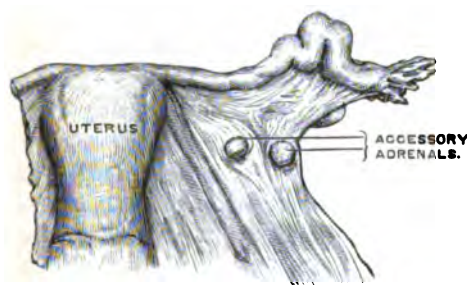


Fig. 3.—Accessory adrenals in the mesometrium of a child. (After Marchand.)

It is a curious fact that accessory adrenals in this situation have only been detected in fœtuses, whereas in the kidney and liver they have been almost exclusively observed in adults.

The similarity of the tissue in some of these renal sarcomata of adults to that of the adrenal is very striking; and though some writers are doubtful of the origin of such tumours from accessory adrenals, nevertheless they form a species with

such peculiar characters, that it should be classed apart from the common variety of renal sarcoma which arises in the connective tissue of the renal sinus.

There are many reasons for encouraging investigations into the histology of malignant tumours of the kidney. Recent writings show clearly enough that they belong to two distinct groups, the sarcomata and the carcinomata.

Renal sarcomata of infant life arise in the connective tissue, which is so abundant in the renal sinus in the earliest years of life. Some examples occurring in adolescents may have a similar origin, but the majority of malignant renal tumours of adult life are either subcapsular tumours accredited to aberrant adrenals, or they are true carcinomata, arising in the uriniferous tubules.

The importance of an inquiry in relation to these three genera of tumours is that it should help us to determine their relative malignancy. Nearly all the cases recorded up to this date, in which the kidney has been successfully removed for malignant tumour, have succumbed within a year from recurrence. In a few rare instances the patients have been reported alive four, five, and six years afterwards. At present there is absolutely no evidence to show whether these survivals were cases of tumours like the one the subject of this record, or carcinomata. It should be borne in mind that this can only be determined by collecting accounts of cases in which the clinical and histological details are given; one without the other is useless.

Ulcer of Stomach; Perforation; Caliotomy.

On May 3rd, 1897, a servant girl æt. 19 was found lying on the floor of a room vomiting violently and in great pain. At 3 o'clock she was admitted into the Middlesex Hospital, where Mr. A. Baldwin and Mr. Goulden found her too ill to give any connected account of the seizure, but elicited the statement that she had for a long period suffered from pain in the stomach after food. On examining the belly they found no obvious distension, but the usual area of liver dulness was replaced by resonance. On this they came to the conclusion that the symptoms were due to perforation of a gastric ulcer. With this view I concurred, and at 6 o'clock I opened the abdomen by an incision in the linea alba, midway between the tip

of the sternum and the navel. On incising the peritoneum some gas escaped. The anterior wall of the stomach was healthy, but a collection of green fluid caught the eye lying between the layers of the great omentum. The omentum was torn through to afford access to the posterior wall of the stomach and the lesser bag of the peritoneum. After a little search my finger slipped through a circular hole about two centimetres in diameter, with rounded edges, situated on the posterior wall of the stomach near the cardiac orifice; the edges of the ulcer were conjoined by a thin continuous silk suture; the peritoneal and muscular coats adjacent to the ulcer were then brought together by another continuous silk suture in such a manner as to turn the edges of the ulcer into the stomach; here and there an interrupted silk suture was added for greater security. The incision in the belly wall was then prolonged some distance below the navel, and the belly cavity thoroughly irrigated with warm water (105° F.). Every viscus was thoroughly sponged, and a large rubber drain introduced into the lesser bag of the peritoneum and the incision closed. Silkworm-gut sutures were employed to secure the muscles, fascia, and peritoneum; the skin was secured by a continuous suture of fine silk. For a few days she was maintained by nutrient enemata, but allowed to sip water. On the third day milk and soda water were allowed by the mouth, and on the sixth day this was increased to two pints in the twenty-four hours.

The employment of a drain-tube necessitated the daily dressing of the wound. On the 16th (twelve days after the operation) the food was thickened with arrowroot, custard pudding was allowed, and pounded chicken. At this date the temperature, which had been gradually rising, reached 103° Fahr. The pulse remained at 80 per minute, and of good volume. On the 20th the temperature fell in a few hours to normal, and remained so for eight days, then it began to rise again. All solid food was stopped, and the temperature soon became normal.

On May 22nd the drain-tube, which had been gradually shortened, was discarded, and on June 10th the girl left the hospital for a convalescent home with her wound soundly healed. She reported herself three months later in excellent health.

Pyloric Obstruction due to a Band; Coeliotomy; Recovery.

In April of this year Dr. Fenton asked me to take charge of a patient in the Chelsea Hospital for Women, who was complaining of great pain in the region of the gall-bladder, distension of the stomach, and vomiting.

The woman was æt. 41, and well nourished. When attacked by pain a distinct sausage-shaped tumour could be made out in the right hypochondrium, immediately below the costal arch; directly over the swelling there was a vertical linear scar 5 cm. (2 inches) long, the result of a coeliotomy performed at a provincial hospital.

The vomited matter consisted of usual gastric matters, free from blood or sarcinæ. A careful consideration of the clinical features of the case induced us to advise the patient to submit to operation. We were of opinion that the signs, though indicating the impaction of a biliary calculus in the cystic duct, might perhaps be due to obstruction of the pylorus, but the general condition of the woman enabled us to exclude carcinoma of the stomach.

She willingly submitted to our proposals with the hope of obtaining freedom from pain.

On April 3rd the parts were exposed by a vertical incision on the inner side of and parallel with the old cicatrix. The gall-bladder and liver were healthy, and the ducts free from obstruction. On endeavouring to draw the pylorus into the wound it was found to be restrained by a narrow fibrous band passing across it from the lesser to the greater omentum. The stomach was greatly distended. On dividing the band gas at once gurgled through the pylorus into the duodenum, and the stomach collapsed like a punctured gas-bag. The parietal wound was sutured with fishing-gut, and dressed with gauze.

The relief was almost magical. The gastric disturbance and pain ceased; the wound healed quickly, and the patient returned to her home April 27th.

I am unable to offer any suggestion as to the nature of the band. I have not seen anything like it in relation with the stomach, though such bands are common enough in relation with the small intestines.

A Tumour (Adenoma) of the Pancreas.

In May, 1897, Dr. Hollings placed under my care a single lady, æt. 28, on account of a large lump in the belly presenting many of the signs common to a solid tumour of the left ovary. The patient had been conscious of a local enlargement for about a year, but during the past three months it had greatly increased in size, and had caused much inconvenience and a sensation of dragging; moreover, she had become very thin. She was admitted into the Chelsea Hospital for Women, and observations made on the quantity and quality of the urine, for in some points the swelling resembled a renal tumour. It was finally decided to perform cœliotomy. On June 8th I explored the abdomen through an incision in the linea alba below the umbilicus. It was at once discovered that the tumour was retro-peritoneal. The median incision was closed, and the tumour exposed through a long opening in the left linea semilunaris. The mass presented to the inner side of the descending colon, and the peritoneum was incised over the most prominent part of the tumour, and I proceeded to enucleate the mass. This caused an excessively free venous hæmorrhage; the capsule contained veins of large size and in great number. At length the tumour was shelled out of its bed, save its pedicle, which lay exactly over the spine. The pedicle was transfixed and secured with silk ligatures. The large veins in the capsule were secured with thin silk ligatures, and in order to control the bleeding from a large vein near the pedicle it was necessary to bunch up and transfix the sac. With a thin silk suture I secured the free margin of the sac to the peritoneum, and stuffed it with iodoform gauze. The patient now became so collapsed that she seemed about to die. Whilst I sutured the wound, preparations were made to perform transfusion.

As soon as the wound was secure I opened the right median basilic vein and introduced two pints of warm saline solution. The result was excellent. The pulse regained its volume, and the patient was returned to bed. In a few hours the shock passed off; patient retained milk and soda water, and had some sleep. In forty-eight hours the gauze plug was removed, and a drain-tube substituted. In four weeks she went to the convalescent home. Whilst there some of the ligatures came away, and she rapidly regained strength.

The tumour is in many respects remarkable; it is ovoid, measuring 15 cm. in its long axis. The capsule is thick, spongy, and vascular. On section it is made up of a collection of isolated loculi filled with soft, reddish, friable tissue. The soft material (examined microscopically) is composed of tissue exhibiting the structure of normal pancreas.

Punctured Wound of the Liver from a Railing Spike.

On August 2nd, 1897, a boy 9 years of age, was creeping along the top of a gate armed with sharp iron spikes 10 cm. in length, after a cat; he slipped, and a spike entered the right groin. He was ultimately conveyed in a collapsed condition to the Middlesex Hospital, and the house surgeon, Mr. Mudd, noticed a small piece of omentum hanging from the wound. I had the boy placed under ether, and found a wound 3 cm. broad situated 2 cm. to the inner side, and the same distance below the anterior superior spine of the right ilium. From this opening a piece of omentum protruded, and the tissues in the line of Poupart's ligament were emphysematous. The finger easily passed above Poupart's ligament, and then entered a hole in the abdominal wall immediately over the cæcum. An incision was made in the belly exactly on the lines adopted for exploring the vermiform appendix; on incising the peritoneum much free blood escaped from the cœlom (general peritoneal cavity): the protruding omentum was ligatured with thin silk and removed. I then perceived that there had been a free extravasation of blood into the belly, and as the iliac arteries were uninjured, I suspected that the liver was probably its source. On enlarging the incision to expose the under surface of the liver a triangular rent was seen in the right lobe, with a deeper perforation near its centre. Pressure was applied to the rent by means of hot sponges, and some ounces of blood were removed from the pelvis. A careful examination of the adjacent coils of gut led to the detection of a star-shaped split in the peritoneal coat of the ascending colon, but it did not penetrate the whole thickness of the muscular coat. The serous and muscular layers of the abdominal wall were secured with thin silkworm-gut sutures. Before securing the sutures a padding of iodoform gauze was adjusted to the under surface of the liver, and its end drawn into the wound. The

sutures were secured, and the skin brought together with a continuous silk suture.

The boy quickly rallied, retained milk and soda water, and slept well. Forty-eight hours later he was again chloroformed; the upper angle of the wound was reopened, and the gauze plug withdrawn, and a very narrow drain-tube substituted. This was withdrawn on the third day. The boy left the hospital convalescent on the fifteenth day.

SOME CASES OF INTEREST IN MAMMARY DISEASE,

With Remarks.

By A. MARMADUKE SHEILD, M.B., F.R.C.S.

PART II.

Lactation eczema.—Three cases of this troublesome affection have come before my notice in the last six months, and they all had certain symptoms in common. The women were young and delicate looking, with fair skins and blue eyes, and had suffered from cracks and fissures about the nipple, which had been congenitally retracted and difficult for the infant to seize. I have no doubt that the proper care and management of the nipples in growing girls is a matter too much neglected by parents and family practitioners, and that the pressure of tight corsets has much to do with the flattened condition of these very important organs. It seems clear that cases of eczema about the nipple are infective—that organisms gain access to the lymphatics of the skin by minute abrasions, from the infant's mouth, or the moist sodden surface about the areola. In cultivations of scrapings about the skin of the areola the *Staphylococcus aureus* was found in abundance. Organisms in the mouth of the infant are very abundant, and probably more than one kind may aid in producing the malady. The symptoms are quite typical; the painful cracks and fissures, the exudation drying with glazed scales, the red and hyperæmic appearance, are all characteristic of the malady. Should the affection become chronic, the surface presents a brownish aspect, and the skin becomes definitely thickened. In these cases confusion with "malignant dermatitis" is readily

fallen into, and I doubt not that cases of "malignant dermatitis" cured by external remedies have been really instances of mistaken diagnosis. The principal distinctions are as follows:—(1) True eczema takes its origin usually in lactation, and is found in young women. (2) The surface lacks the vivid red, raw appearance of malignant dermatitis. (3) Scrapings of eczema fail to show the large vacuolated cells so familiar in malignant dermatitis (so-called psorosperms). (4) Even chronic eczema is more rapid in its history than true malignant dermatitis, which is essentially a chronic malady. (5) Eczema has not the sharply-defined border of "malignant dermatitis," and the thickening of the integument is not so clear and definite. The appearance to the eye is perhaps the most important distinguishing sign, for the colour of "Paget's disease" is typical and utterly unlike anything else—when once seen it is never to be forgotten. In cases of doubt, occurring in older women, the rebellious behaviour of malignant dermatitis to all remedies is a significant sign of its true nature.

It may be remembered that a small patch of eczema with cracks or fissures near the nipple may be the starting-point for an outbreak of general erysipelas. I have seen this occur on several occasions. The erysipelas has been of the variety to which the term "wandering" used to be applied. The constitutional symptoms were severe, and the "blush" and œdema occurred in various parts of the skin—as the chest, arms, and forehead.

Another most important reflection regarding eczematous cracks and fissures about the nipple is the possibility of confounding such conditions with syphilitic infection. Typical chancres about the areola will probably be easily recognised. The lesion of syphilis is, however, not always typical, and, as on the penis, a very insignificant and easily overlooked lesion may prove to be true syphilitic infection. In cases of suspicion it is well to wean the child and suspend judgment, keeping a careful watch on the axillary glands and waiting for skin manifestations to arise. "Secondary" syphilitic rashes about the areola are also readily mistaken for eczema. The brownish coppery tint and tendency to serpiginous formation should at once arouse suspicion.

As regards the treatment of lactation eczema, the local applications advised are almost endless.

The child had better be weaned, and should there be much hyperæmia and inflammation, soothing remedies should always be applied. Calamine lotion, the "lotio lactis," and rice water are all excellent. Bearing in mind the bacterial nature of the disease, mercurial applications are generally advisable, and these should be at first dilute, gradually increasing their strength. Unna's plasters containing such drugs as boric acid or ichthyol are very valuable, and the latter drug has given me excellent results in eczema about the nipples. On the more chronic cases, nitrate of silver may be applied, to be followed by mercurials. Weak tar applications are also useful, with salves or plasters of salicylic acid or resorcin; but these latter remedies must be employed with caution, and very dilute at first.

All possible means of improving the general health must be taken into account, and iron is the drug which seems to act most beneficially. It must be given in a mild form, or as "Levico water," which seldom constipates or causes digestive disturbance.

Cysts of the breast.—It is often stated that cysts of the mamma are comparatively uncommon. A number of these cases remain untreated, and many are never admitted to hospitals; I believe, however, that they form a far larger proportion of cases of mammary disease than is generally believed. The following cases of interest have lately come under notice.

A lady aged between 40 and 50 saw me in the spring of this year for a tumour of the breast, which she had noticed for about three months. It was deep in the mamma, and felt about the size of a small hen's egg, very hard and fixed. The nipple was retracted, but this had always been the case, and the skin over the swelling was not implicated. The axillary glands were apparently not enlarged; no nipple discharges had been noted. As is usual in such cases, the most varied opinions had been expressed, but the diagnosis of cancer had been confidently given. Singularly enough, about twelve years ago this lady had been pronounced to be suffering from incurable carcinoma of the cervix uteri, by some of the most distinguished authorities of the day; all symptoms of this had now disappeared, and the cervix had returned to a normal condition, but I was unable to verify this by examination. As all operative

interference was at first declined, I placed a mercurial plaster over the part, with a layer of cotton-wool and a firm bandage.

In three weeks' time I again inspected the case. The tumour was there as usual, but it had changed its characteristics, being distinctly less hard and prominent, with a dim sense of elasticity on firm pressure with the pulp of the index finger. Finally an exploratory incision was permitted. A considerable depth of breast tissue was cut through gradually, the parts being well held asunder and sponged. The formation, whatever it was, lay almost on the pectoral fascia. Even when closely approached it was impossible with the finger to be sure of its true consistence. In cutting into it, it proved to be a cyst with slightly coloured mucoid contents and thick walls, the size of a walnut, its parietes smooth internally, and devoid of any intracystic growth. The cyst, with a liberal margin of mammary tissue, was excised, and a drainage-tube brought out inferiorly; the deep wound was united with deep and superficial sutures, and soon healed. Under the microscope the wall of the cyst was lined by smooth endothelium. The mammary substance around was thickened, and in a state of chronic inflammation.

This case bears its own lesson. I am confident that such instances are more common in practice than is generally believed. Gross has made the significant statement that the majority of cysts of the breast occurring in elderly women are only discovered after removal of the entire organ under the supposition that cancer existed. This statement would, perhaps, hardly hold good now, yet it is surprising how little the close similarity between a deeply-seated cyst and a cancerous tumour is recognised. Exploratory incision is the one method by which mistake can be avoided. The remarkable series of cases published by Bryant in vol. xix of the 'Medical Society's Transactions' are worthy of attention in this respect, and it is right to state that to this surgeon the teaching of the value of exploratory incision is largely due.

On April 2nd, 1897, I saw by the kind advice of Dr. Meckay, of Devizes, a lady of middle age, a widow who had a family, and who had always been able to suckle without inconvenience. She was well and healthy, but complained that a discharge constantly issued from the left nipple and soaked her linen. This had been going on for at least

five years, and she gave a history of a swelling being found occasionally, which she had shown to a hospital surgeon, who advised that nothing should be done. On examination the breasts felt normal, with one exception. On the left or affected side the nipple was comparatively retracted, and to its inner side was an indistinct swelling the size of a hazel nut, which felt like a tumour or circumscribed inflammatory patch. On firm pressure with the finger upon this, a droplet of yellowish fluid exuded from the nipple. I advised exploratory incision, with permission to remove the whole mamma should growth be found. This was, however, refused, and the patient insisted at all hazards upon the mere removal of the local trouble, whatever was revealed by incision. The operation was done on April 6th, and a cyst with yellow glairy contents, the size of a small cherry, was opened. In close juxtaposition lay another, the size of a pea. On the wall of the larger cyst was a stalked papillomatous growth the size of a pea. The cysts, with the portions of mammary tissue round them, were freely excised, and the rather deep wound united by deep and superficial sutures, with a horse-hair drain inserted into the depths of the cavity; sound healing took place within a week. Examination with the microscope showed that the intra-cystic growth was entirely confined within the cyst; it was dendritic, and the central stalk covered with large cells. There was no evidence of malignant action. The specimen falls into the class of duct papillomatous cysts described in the 'Clinical Journal' December 16th, 1896.

Remarks.—The treatment of cysts within the breast associated with intra-cystic growth is fraught with difficulty, and differences of opinion must arise. It is seldom that the remainder of the mamma is healthy in these cases, and fresh cysts are very apt to form in other parts of the breast should the organ be left.

Again, without the microscope, and even with it, it is always difficult to declare with confidence whether an intra-cystic growth is innocent or not. Once the villous protrusions and the epithelium with which they are covered transgress their confines and infiltrate or extend into the adjacent tissues, the villous variety of duct carcinoma is apt to originate.

In women of middle life, therefore, it would seem safer to remove the whole mamma for cysts

which contain intra-cystic growth in any abundance, especially if the walls of the cyst are at the same time thickened. The following case further illustrates this most important and very difficult practical subject.

In July, 1897, I saw a married woman aged thirty-nine, a patient of Dr. Wynne, of Limpsfield, who was childless, and who had long noticed deep retraction of the left nipple. In fact, the nipple had been "contracted as long as she could remember." Within the last two years she has suffered occasional attacks of pain, slight bleedings, and the escape of a yellow and viscid discharge. On examination the nipple was deeply retracted so as to resemble a deep, circular, crateriform depression. At the bottom of this was a florid red, moist, granular-looking growth, exactly resembling the "vascular tumour" of the umbilicus of children. Continuous with the nipple and stretching towards the axilla across the areola was an ill-defined tumour, evidently composed of altered breast tissue. It was firm, lobulated, and felt exactly like an area of chronic mastitis, except that here and there slight prominences, presumably cysts, were perceptible to the fingers. The axilla was quite free. The remainder of the mamma and the opposite breast were apparently healthy.

An exploratory incision was made on July 24th. The cut area was tough and resistant, and a piece of breast tissue the size of a dessert-spoon was literally honeycombed with cysts. Many of these were fusiform in shape, and obviously dilated larger ducts. Others were rounded, and in size all varieties could be seen from a pea or currant to a small shot. They were full of glairy yellow fluid. Some of them contained intra-cystic growths attached to a tiny stalk, so fragile that the growths fell out of the cysts on washing the specimen; a larger and more vascular growth sprouted out of a dilated duct into the floor of the umbilicated depression, it spread out in its interior, and quite filled it. Seeing the dubious nature of these growths and the age of the patient, the whole breast and axillary fascia and lymph-glands were removed. The remainder of the mamma seemed healthy; an occasional small cyst was seen here and there. The accompanying sketch well illustrates the appearances; it gives no idea of the enormous number of cysts, which were only seen at the moment of division with the scalpel. Under the

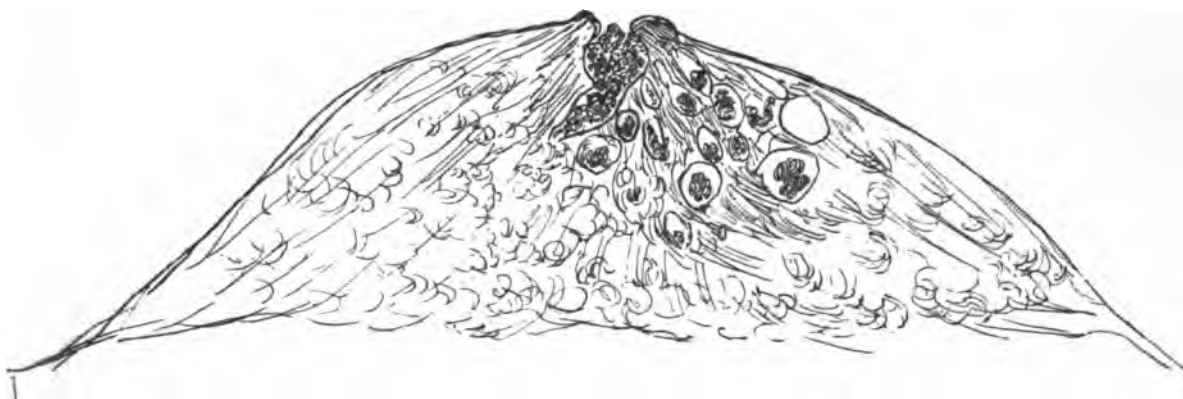
microscope Dr. Rolleston found that the papillomatous growths were of the usual structure, with no trace of carcinomatous degeneration about them. The removal of the mamma was justified from the extent of the disease, but the growths seemed of an innocent nature, and definite cure is to be hoped for.

This case is a very unusual one, from the limits of the morbid process to the larger ducts near the nipple, and the temptation to assume a parasitic cause for the growths within the dilated ducts is very great. One can hardly doubt that were such a case left, the growths would increase, and take on malignant action, bursting through the cyst walls and infiltrating the mamma. Removal of the entire breast is wise in cases like this one where such growths are very abundant.

Operations on carcinoma in old age.—It occa-

a hard dry scab. The axillary glands are little, if at all, affected. In such cases the patient and the carcinoma gradually decline together, and the growth seldom causes such local trouble as to justify the advising of an operation. The following case illustrates well some of the preceding remarks.

In May, 1897, I saw a lady, æt. 83, with an undoubted carcinoma of the breast. She had nipple trouble for many years, a moist discharge with irritation and retraction. The tumour had only been noticed for a few months, but it may have existed longer. It was about the size of a Tangerine orange, very hard, heavy, and not at all tender or sensitive. The skin was adherent over it at one spot by a slight dimple. The axillary glands were not affected. The old lady was healthy and vigorous for her age, and did not



sionally happens that carcinoma attacks or is found in the mammae of extremely old persons. Through the kindness of Mr. J. R. Lunn, F.R.C.S., I saw a male patient last year who had carcinoma of the mamma at the age of 91. There is now or was lately a woman under his care who had a growth almost certainly carcinomatous in the mamma. She is reputed to be 100 years old! These are extreme instances, but cases between seventy and eighty are not so very uncommon. At such ages the advisability of operation is always a difficult one to determine. Much will depend upon the nature of the growth, as to progress, tendency to fungation, and especially as to whether it causes pain and distress. In many aged persons the tumour is small, shrivelled, withered, and atrophic. If it implicates the skin it only shows a small superficial ulcer covered by

suffer from the presence of the growth. As is usual in such cases, there were differences of opinion as to whether operation should be performed or not. I counselled against for the following reasons.

1. The growth was chronic, and at her extreme age would probably not quickly advance.
2. It caused no local suffering or inconvenience.
3. The administration of an anæsthetic, removal of the mamma and axillary lymphatics, with the confinement to bed for even a few days, would be likely to prove risky in so aged a subject.

The risks would be bronchitis or bronchopneumonia with pulmonary œdema, or that general failure of the vital powers which sometimes ensues in the aged from slight injury or disease. Thus the surgeon might shorten life in removing a local trouble not likely to cause great

discomfort or even to curtail the term of the individual's existence.

Question of operation on recurrent carcinoma.—There are few cases which give rise to greater differences of opinion than these. As a general rule, local recurrences which do not involve the axillary vessels and nerves should be removed, and that repeatedly. Cases where life has been longest preserved after carcinoma of the mamma have been those where strictly local recurrences have been removed in the form of small, button-like deposits in the scar or its immediate neighbourhood. Extensive infection of the skin, either in the form of multiple tubercles or infiltration, forbids fresh operation, and so, as a rule, does evidence of deposits of cancer in the spine or viscera. A most important point is the condition of the cervical glands at the root of the neck. Hard fixed glands here generally mean infection beyond the reach of operation. It may be thought that the detection of these would be simple enough, but in practice we meet with strange difficulties in the diagnosis of hard tumours in the subclavian triangle.

On August 3rd, 1897, I saw a patient, æt. 53, under the care of Dr. Codd, of Bromley, and her right mamma had been removed for carcinoma five months before. The incision "tailed off" into the axilla, but hardly seemed free enough in this direction to ensure that a thorough removal of the axillary lymphatics had been effected at the primary operation. There was now, on the inner wall of the axilla, a recurrent mass of growth the size of a walnut, but movable, and obviously within the reach of operation. At the right root of the neck, deeply seated behind and above the sterno-clavicular joint, was a hard lump the size of a hazel-nut, only to be felt on deep and careful palpation; it felt so hard and fixed that I concluded it was bony, and at first expressed an opinion that it was an enlarged tumour process of a cervical vertebra, which I have known more than once simulate enlarged glands in this situation. The patient was lying down, and the tissues of the neck were tense. On making her sit up and bow the head forward, it could be appreciated that the hard, deep little tumour moved ever so little from side to side. It was thus too certain that a cancerous gland here existed, and no operation was therefore advised. I doubt if it would

have been possible to remove this little growth, since the pleura and subclavian vein would have been found adherent to it. Even if such a mass could be safely taken out, other glands would remain undetected at the time of operation, but soon to become prominent after it. The only operation feasible in such cases is removal of the pectorals and division of the clavicle, the glands being removed from the axillary sheath right into the root of the neck. Most surgeons would long hesitate before proposing so serious an operation for a problematical result.

Among the more common conditions which closely simulate serious tumours of the breast, abnormalities or deformities of the ribs hold a prominent position. This may be the result of fracture, but more commonly one or more ribs are curiously bent near their sternal ends, leading to an obtuse prominence, visible to the eye and perceptible to the touch, and apparently becoming more prominent as growth of the thorax is more advanced—towards the age of sixteen or twenty. This curious deformity is noticed by accident in connection with some slight neuralgic pain or from an accidental blow, to which all the troubles are attributed. Its discovery often causes the greatest alarm. The following is a marked example of this condition, very important in practice.

An unmarried girl æt. 23 saw me at St. George's Hospital on August 18th, 1897. She gave the history of being struck with a golf ball on the right mamma about three or four years ago. It was doubtful whether this had anything to do with the present trouble. About two months ago she noticed a swelling in the right breast quite by accident. The patient is a healthy-looking subject. On examination a prominent tumour is seen in the upper part of the right mamma. On examination this is produced by the third, fourth, and fifth ribs, which are acutely flexed just anterior to the angles, so that an obtuse ridge-like prominence is formed, pushing the mamma forward upon it. The breast itself is quite healthy, and no true tumour exists.

The difficulty and doubt which surround the diagnosis of some inflammatory conditions of the mamma is well illustrated by the following case. A married woman æt. 62 was admitted into St. George's Hospital on August 9th, 1897. She has felt a painful swelling in the left breast towards its axillary aspect for about one month. It has

rapidly increased, and is associated with "shooting" pains. She has suckled with the affected side, and there has never been trouble with the breast before.

On examination the woman is pale and thin. There is a nodular, lobulated, hard, fixed tumour, the size of an orange, in the left axillary wall, which feels as though it were in the axillary part of the mamma. The skin is adherent and slightly red, and the swelling is tender on palpation. Beneath the left clavicle there is a marked fulness; the cutaneous veins are enlarged. There is nothing abnormal in the condition of the nipple. There was slight increase of local heat, but no elevation of temperature. The diagnosis of carcinoma had been positively given; indeed, the case resembled it closely.

Spirit lotion was applied, and iodide of potassium administered internally. The case was watched for a time. By August 24th the diagnosis of fluid was clear. The formidable hardness of the tumour had given way to a feeling of fluctuation and yielding at one spot, where the skin was red and thinned. An incision revealed a large abscess, which seemed to have originated beneath the pectoral in the lower anterior lymph glands. The mammary substance was implicated, but the abscess had not originated there. The fulness beneath the clavicle was due to the fact that the pus had burrowed in that direction. After drainage the progress of the case was satisfactory.

This case serves as a text for the consideration of one of the most important matters in the diagnosis of mammary affections, *i. e.* the detection of chronic abscess of the breast. In acute abscess the symptoms are generally typical enough. The only possible error here is the confusing acute abscess and inflamed soft carcinoma with suppuration within a cyst. The latter condition is fortunately very rare.

Abscess of a chronic nature forming slowly in the breast of an elderly woman is exceedingly like a solid tumour, because, as in the present instance, the surrounding tissues are thickened and greatly indurated, and the actual inspissated pus may be surrounded by a dense and thick wall.

Moreover, the nipple is commonly enough retracted from some old inflammatory attack, and here in a thin and pale grey-haired woman we have the picture of cancer complete.

The distinguishing diagnostic signs are not many or reliable, but by observing the following points a shrewd guess may be formed of the true nature of the malady even before incision.

1. Early cancer is seldom painful, "throbbing," "shooting" pains belong to inflammatory affections, and local tenderness is very strong evidence of inflammatory origin. Early carcinomata, if not artificially inflamed by excessive handling, rubbing with liniments, &c., are seldom painful and tender on manipulation. In the case just related the tenderness on palpation was very marked, and at once made me suspicious of the true nature of the affection.

2. In chronic mammary abscess evidence of bygone suppurations are not uncommon. Scars may mark the site of the openings of an old "lactation abscess." I feel very certain that a certain number of chronic mammary abscesses arise in the site of the old inflammatory foci of antecedent suppurations.

3. In the very chronic cases there is no rise of temperature or increase of local heat, and it must ever be remembered that rapidly growing malignant growths may be associated with fever of the "hectic type." The temperature chart is not therefore invariably to be relied upon as a guide between suppurative affections and growths.

4. In a considerable number of instances firm pressure with the pulp of the index finger upon the centre of the swelling will detect a sensation of yielding or elasticity. This is in direct opposition to what is found in hard carcinoma, when the centre is usually the most hard and "stony" point. I regard this sensation of yielding on pressure as being the most important local symptom of chronic abscess.

5. In a certain number of cases, exploratory incision is the only certain method of distinguishing between a chronic abscess and a deeply-seated nodule of cancer, and this should always be done before removing the breast. Some authorities assert that in cases of chronic abscess it is well to remove the mamma. The woman is of advanced age, the abscess and inflammatory action have damaged the mammary tissues, and cancer may then originate. In commenting upon this attitude I may point out that removal of the mamma is always looked upon with great repugnance by patients, and that abscess in elderly

women is by no means universally followed by carcinoma. I cannot but think that the custom of removing the breast for chronic abscess has originated in an attempt to escape from operative error. If these abscesses are drained inferiorly, their walls curetted and flushed, and methodical bandaging afterwards applied, the final result is very good. Amputation should never be performed unless the mammary tissue is riddled with sinuses, and there be strong presumption of tubercular origin.

CLINICAL LECTURES ON URINE.

Delivered at University College Hospital by

J. ROSE BRADFORD, M.D., F.R.S., F.R.C.P.,

Physician to University College Hospital.

VIII.

Salts of the urine.—The most important of the urinary salts are the chlorides, the sulphates and the phosphates; the urates we have already considered, the other salts are not of any very great practical importance, with the exception perhaps of the oxalates. There are minute traces of other salts present, but they are not of sufficient importance to detain us. The salts of most clinical importance are the phosphates and the oxalates; the chlorides and the sulphates are not of any great clinical interest, and this perhaps more especially applies to the sulphates. The chlorides are very abundant, and are readily detected, but the physiology and the pathology of the chlorides is very little understood. The bulk of the chlorides in the urine is derived from the chlorides of the food, but not entirely so; and the chlorides undergo in disease, more particularly in febrile diseases, changes leading to a very great diminution in the amount excreted, and that is not altogether a question of diet. It used to be taught that pneumonia was characterised by diminution in the chlorides in the urine, but it is not characteristic of pneumonia, it is really more characteristic of a high temperature. A patient with pneumonia has a very remarkable temperature chart; the temperature will reach 104° or 105° in under twenty-four hours, and it will remain at this height, with very slight oscillations of perhaps not

more than one degree, and under these circumstances the chlorides undergo a noticeable diminution. Tonsillitis, especially some of the varieties known as hospital throats, is also characterised very often by high temperature— 105° is not an uncommon temperature, and it is not at all uncommon to get great diminution in the chlorides in this disease. Pneumonia is perhaps a little peculiar, because the diminution is greater in amount than in the other febrile processes, but this diminution is not characteristic of pneumonia. It used to be said when I was a student that it was one way of diagnosing pneumonia; in pneumonia the physical signs may not be shown for four or five days, therefore you may be in some doubt as to the nature of the febrile illness, and it was thought that the examination of the urine afforded a means of confirming the suspicion you might form with regard to the nature of the disease. You took the suspected urine, and you also took a normal urine, equal amounts in two test-tubes, and you took a stick of nitrate of silver and dipped it in both. In one you would notice a dense, very flocculent precipitate; in the other you would have a faint cloudiness only. But this is not of sufficient importance to detain us long. There is one point which is of importance; there is a great deal of evidence that if you have a diet which is deficient in chlorides, and hence the urine is scanty in saline ingredients, the uric acid is liable to be precipitated, and that is perhaps the most important point about the chlorides in the urine. A high percentage of salts, not limited to the chlorides, is necessary in order to keep the uric acid normal in the form of quadriurates, and a diet scanty in saline ingredients is held to be a predisposing cause in causing uric acid calculi. It has long been known that stone is common in India, and the same remark applies to certain parts of China; at least, so it is said. In both of these countries the formation of the uric acid diathesis cannot be held to be due to an excess of nitrogenous food, because in India the diet is largely carbohydrate, and it has been shown that such a diet as this contains relatively small amounts of saline ingredients. The same thing has long puzzled people in this country. It is the experience of most lithotomists that more stone appears in children in the hospitals than in private practice; and Sir Henry Thompson, who had an

almost unrivalled experience in the matter of stone, had only seen very few cases of stone in children in his private practice. Children in private practice are more likely to be better fed, hence it has been a puzzle why stone is more common in the hospital patients who are poorly fed, and the same explanation has been held; these children live on tea and bread and butter; in London they do not get much milk, and not much in the way of proteid food, tea and bread are the staple articles of diet, and the amount of saline material ingested is scanty; and I pointed out to you when discussing uric acid, that a meat diet is not only rich in nitrogenous material, but is rich also in saline material, both phosphates and chlorides, so that the chlorides may undoubtedly play an important part indirectly in that way, in keeping up a high percentage of saline material in the urine, and in that way keeping the urates in solution.

One does not know any sound explanation of the reason for the diminution or even the disappearance of the chlorides in the urine in febrile states. There is no satisfactory explanation, and I do not waste your time over the cut and dried explanation which says that the chlorides are not in the urine, because they are in the exudation in the lungs in pneumonia. In fact, there is really no satisfactory explanation to be offered. The sulphates, I think I may say, are of practically no clinical importance, but there is a good deal of scientific interest about them. Sulphur in the urine, as you probably know, is excreted in three forms: first of all as ordinary sulphates, metallic sulphates; secondly, it is excreted in the form of what are known as aromatic sulphates, compounds of sulphuric acid, with various aromatic bodies of the indol and skatol group,—a considerable amount of sulphur is excreted in that way; and lastly, a small amount of sulphur is excreted in the urine normally in the form of taurin-like bodies, and the only interest of that is in regard to the relationship of the taurin group to the cystin group. There is a rare calculus formed of cystin, and cystin is altogether an abnormal substance, and is rarely met with, but it is really allied to a normal constituent of the urine, viz. the sulphur compounds of the taurin type. The sulphur of the urine, like the chlorine and phosphorus in the urine, is derived partly from the sulphur of the food and partly from the sulphur of the body, and the sulphur of

the body is derived from the proteids, and hence it has been supposed that the excretion of the compounds of sulphur will be modified in conditions in which there is extensive wasting of the proteids of the body. The sulphur of the food is largely excreted as ordinary sulphates; the sulphur of the body, on the other hand, is excreted probably very little as sulphates, but largely as aromatic sulphates, and hence at one time it was supposed that the aromatic sulphates gave a clue, or index, or measure to the amount of waste of the proteid tissues of the body; but it was found that the aromatic sulphates, although in part formed no doubt by the waste proteid tissues of the body, were largely formed in the alimentary canal as a result of the putrefactive processes carried on there, or the decomposition, if you like, occurring in the alimentary canal during the progress of digestion according to some physiologists; or according to others this decomposition is due to the activity of the microbes normally found in the intestines. There is a considerable amount of decomposition in the alimentary canal, and the aromatic sulphates are formed here. It was supposed they were formed from the proteids of the body because it was observed they were present in the urine even during starvation, and hence the idea that they were formed from the tissue proteids. It was shown by a man who died a short time ago, Baumann of Freiburg, that in starvation you get what are called artificial fæces; you have an exudation into the lumen of the intestine from the blood-vessels of the intestine, and this exudation contains proteid matter, and therefore it is quite possible that even the aromatic sulphates of starvation have largely an intestinal origin. These aromatic sulphates are undoubtedly formed in the intestine, even when no food is taken, but it is possible that they are also formed by the breaking down of the tissues in the body at large; and of course there is a well-known physiological fact, a well-known experiment of Bunge, that animals fed on proteids deprived of salts (mice fed on casein, having had the lime washed out of it) will die more quickly than if they are starved. Death occurs sooner than if they are starved when they are given food without salts; and Bunge held the view, and adduced evidence in favour of the view, that this was due very largely to the formation of sulphuric acid with no inorganic material present

to neutralise this powerful acid. It is definitely proved that aromatic sulphates are formed in the intestine, and it is probable that they are formed by the breaking down of the proteid tissue in the body; it cannot be said that they are formed away from the intestine; it is possible that even in starvation the exudation in the intestine (the artificial fæces) is responsible for the formation of these aromatic sulphates. In health, aromatic sulphates exist in about the proportion of 1 to 12; that is to say, of every 13 parts of sulphuric acid in the urine about 12 parts are excreted as ordinary sulphates, and about 1 part is excreted as an aromatic sulphate. Personally, I always impress upon you the point that the excretion of these aromatic sulphates in the urine is a very curious phenomenon, and shows the relation of the kidney to the alimentary canal. You might have thought that these bodies would have been evacuated by the rectum, instead of that they are re-absorbed and excreted by the urine. The kidney actually excretes material formed in the intestine and absorbed from the intestine, and therefore when the function of the kidney is compromised and excretion is impossible, it is quite possible that the patient will succumb to intestinal poisoning. That is a theory that has been held as regards uræmia, and it is a theory that you cannot dismiss in a few words, because normally it is evident that the kidney does do such work. The amount of aromatic sulphates is a measure of the amount of intestinal putrefaction and of tissue disintegration; and the former is more important because the latter, as I have hinted, is by no means so conclusively proved as the former.

In all conditions in which intestinal putrefaction is increased the aromatic sulphates are increased; hence in all cases of intestinal obstruction or even of constipation, anything from constipation to complete obstruction, the aromatic sulphates are increased, as is also the case where there is extensive intestinal putrefaction and diarrhœa. The aromatic sulphates are also increased in peritonitis where the bowel is paralysed, and you get, as is well known, an accumulation in the lumen of the bowel of a most offensive character, a thin brownish fluid, the exact nature of which is not understood; certainly it is not simply fæcal. The urine in those cases is dark in colour from skatol and indoxyl-ments; and, speaking broadly, the aromatic sul-

phates are increased in all conditions where these pigments are present in the urine. You estimate these aromatic sulphates in the following way; it is not a matter of very much trouble, or of very much clinical importance. You boil the aromatic sulphates with hydrochloric acid, and they are converted into ordinary sulphates,—that is the first point. The second point is that an aromatic sulphate does not form an insoluble precipitate with chloride of barium; and the estimation is based on these two points. You take 100 c.c. of urine, and you add to that an equal volume of a solution of barium chloride and barium hydrate in the proportion of 66 per cent. of the former to 33 per cent. of the latter, saturated solutions of both salts being used. All the ordinary sulphates are thrown down, and you then filter, and to the filtrate you add some hydrochloric acid and you boil it, and having boiled it smartly for five minutes you then put the boiling liquid in a warm chamber, at a temperature of about 100°, for two hours, and thus the aromatic sulphates are converted into ordinary sulphates and precipitated. To estimate the total sulphates you simply take 50 c.c. of the urine and add hydrochloric acid and boil it, and add some barium chloride, and that throws down the sulphates, and then compare the weights obtained in the two experiments and you get the relation between the ordinary and the aromatic sulphates. In diseased conditions the proportion rises considerably. It is more a method of scientific interest than of practical importance.

THE TREATMENT OF INGROWING AND INGROWN TOE-NAILS.

In the 'New York Medical Journal' of March 20th, 1897, J. L. Andrews contributes an article on this practical subject. He first gives a definition of what we mean by an ingrowing toe-nail, and says it is a condition in which one or both sides of the great toe-nail seem to grow downward and press into the soft parts to an extent sufficient to cause pain on pressure, and more or less discomfort while walking. This condition is considered by some to be always due to a rolling upwards of the soft parts over the side of the nail, rather than an actual deformity of the latter.

As a matter of fact we see both conditions, and it is important to distinguish between them before giving a prognosis or selecting a line of treatment.

Where the soft parts are primarily at fault we may promise our patient a permanent cure, without operation, if our directions are carefully followed. Sometimes it will be only necessary to order a proper shoe. This shoe should have a low, broad heel, to prevent a wedging forward of the foot while walking. The inner side should be straight, to prevent pressure on the corresponding side of the nail. The toe should be sufficiently broad and extend far enough beyond the end of the foot to prevent pressure on the outer side from above and in front. If this is not sufficient, pack a small amount of cotton under the edge of the nail to protect the soft parts, and apply a strip of rubber adhesive plaster diagonally around the toe, in such a manner that the soft parts shall be drawn away from the nail without direct pressure over the latter. For this purpose a semilunar-shaped piece of plaster is often better than a straight strip. It should be applied with the convexity forward, one horn beginning just behind the nail on the affected side. This will allow the belly to catch the offending soft parts, while the rest of the piece is carried around the plantar surface of the toe and over the dorsum, crossing the first end. This dressing should be changed every three or four days.

In the first class, where the nail is really deformed, this plan is of very much less value. But many cases will be held in abeyance if we attend to the shoe, apply a cotton protection under the edge of the nail, encourage it to grow out beyond the soft parts, and keep them clean. However, this will sometimes fail, and the nail will become really ingrown. Neglected cases of both varieties almost invariably reach this stage, and this explains why, in dispensary practice, we nearly always see the latter variety.

Here there is always more or less hyperplasia of the soft parts, which are rolled up over the side of the nail in amount usually proportionate to the length of time the trouble has existed, and the amount of irritation present. Next to the nail there is a mass of granulation, discharging more or less pus. In nearly all these cases our treatment must be operative if we effect a cure, and in all of them it will be found the most humane procedure.

A modification of the method devised by Anger

seems to fulfil the indications more perfectly than any other. He removes a wedge-shaped piece, including the offending side of the nail, all of the granulation tissue, and more or less of the hyperplastic soft parts. This leaves a flap with which to cover the raw surface. Anger secured the flap with adhesive plaster, put the patients to bed for a week, and did not let them walk for ten or twelve days. By the method about to be described the patients walk immediately, do not go to bed, and are generally discharged cured in from ten to fourteen days.

The success and comfort of the operation depend almost entirely upon little points of technique, and the writer gives them in detail.

The toes and foot are first scrubbed with soap and water, followed by a solution of bichloride of mercury (1 : 1000). For anæsthesia, ten to fifteen minims of a 2 per cent. solution of cocaine hydrochlorate is sufficient if distributed in the following manner. Introduce the hypodermic needle from before backward, just under the part of the nail to be removed; as soon as the skin is pierced inject a drop or two, and wait a moment before pushing it further; repeat the process until the root of the nail is reached. Withdraw the needle now, and, beginning near the primary puncture, distribute a few drops in the skin and soft parts along the outer side of the nail, nearly as far backward as the first joint. In this way the only pain felt is while the primary puncture is being made, and even that may be done away with by spraying with ethyl chloride or ether.

Next wind a small rubber tube around the base of the toe, to prevent blood from interfering with our work. Before making an incision swab the whole granular area thoroughly with pure carbolic acid, working it well down under the edge of the nail. Failure to do this will often cause infection and delayed union.

The first incision begins anteriorly somewhat beyond the edge of the nail, and is carried directly backward (separating a sufficient amount of the side of the latter) to a point a fourth of an inch behind the true matrix. This is deepened through the underlying soft parts, well down by the side of the phalanx.

The second incision begins anteriorly at the same point as the first, and curves outward and backward to join the posterior end of the primary

incision. This is deepened through the soft parts around the outside of the nail until we reach the lowest part of the first incision.

Remove the wedge thus set free and search the posterior part of the wound carefully, so that no part of the corner of the nail or matrix shall be left. If we leave a piece of nail, immediate trouble will ensue; if only the matrix is left, another ingrowing nail will make its appearance in a few weeks.

If properly fashioned, the flap which we have will fit the opposite raw surface perfectly, and should be secured by one or two catgut sutures in front of the nail and the same number behind. Before removing the rubber band apply a moderately thick pad of gauze over the flap and bind it tightly to the toe with a few turns of gauze bandage. Now remove the rubber tube and complete the dressing.

The patients walk home; and although there is often some pain the first night, it does not last longer, and they keep about with little discomfort. The dressing should not be changed in less than a week, unless there is some indication, and at the end of ten days or two weeks the cure will usually be complete.

The "Cotting" operation has, perhaps, been more frequently used in this class of cases than any other, and it has two strong points: it is very easy to do, and the ultimate results are nearly always good. In point of time it suffers in comparison, as the healing process requires from three to ten weeks. Another objection to it is the often painful process of changing the dressings every two or three days.

The second class of ingrown toe-nails, fortunately rare, will take but little of our time. Here the suppurative process has existed for a long time, and we have on either side and in front extensive hyperplasia, sometimes enough to almost bury the nail from sight. The nail is also lifted more or less from its bed, sometimes nearly as far back as the true matrix. The suffering is exquisite, and it is often impossible to wear any kind of a shoe.

A flap operation is here out of the question; the proximity of a suppurating surface which we must leave will defeat almost any attempt at primary union that we may make. Instead, we should pare off the hypertrophied parts on either side as in the "Cotting" operation. In front the

part should also be trimmed down to the level of the bed of the nail.

In dealing with the nail itself we should be guided by the conditions present. If it is loosened nearly to the matrix, it would be better to pull it out and pack the socket with gauze; if the latter precaution is not taken the new nail will find contracted quarters when it comes out. Where the anterior and lateral parts of the nail are separated for only a moderate distance, the free part should be trimmed off to prevent the collection of pus. In these cases the healing process usually requires from four to twelve weeks.

Therapeutic Gazette, September, 1897.

NOTES.

Addison's Disease.—In one of the last clinics of the semester Professor Neusser discussed the possibility of diagnosing Addison's disease when the characteristic discoloration of the skin and mucous membranes is absent. While it is an extremely difficult problem, Neusser does not consider it impossible. Several interesting cases, which have come to autopsy during the past year, though with lesions of the supra-renals, and without the pigmentary deposits, seem to indicate the points on which the diagnosis can be based. The last one which occurred was a characteristic one and may be taken as a type. A comparatively young man was brought to the hospital in a state of coma. There was no oedema and no signs of any ever having existed, but there was an extremely high-tension pulse and thickened arteries. There was no albumen, and no casts in his urine. He died without coming out of his lethargy, and the only clinical diagnosis that seemed possible to make was that of Bright's disease. The autopsy showed extensive degeneration of the supra-renals. The changes in the arteries were such as are often found in Addison's disease, and such as Kollisko considers as due to the effect upon the vascular system of the absence of the secretion of the supra-renals in the organism and the consequent interference through the sympathetic with the peripheral vaso-motor mechanism. It is on these changes in the arterial system with the high-tension pulse, or a very striking difference in tension between the peripheral pulse and that in the abdominal aorta, for instance, when other causes may be excluded, that a diagnosis of Addison's disease may be made even in the absence of characteristic pigmentation.—*Medical News (N. Y.)*, September 18th, 1897.

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* Specially reported for The Clinical Journal. Revised by the Author.

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ACUTE ARTHRITIS AND EPIPHYSITIS OF INFANTS AND YOUNG CHILDREN.*

BY

FREDERIC EVE, F.R.C.S.,

Surgeon to the London Hospital, and to the Evelina Hospital for Sick Children.

My attention has recently been re-directed to the subject of acute arthritis of infants by one of those curious "runs" of cases which sometimes occur. Since October last I have had no less than five examples under my care.

This affection is most frequent and most typical in infants under a year old, but practically the same condition is observed in much older children. In the latter an epiphysitis is less likely to cause suppuration of the joint, or if it does so, this occurs later.

The following case may serve as a clinical picture of acute arthritis. Case 1.—Sarah R—, æt. 10 months, was admitted with the right knee much swollen, flexed to a right angle, and with deep fluctuation, evidently due to fluid in the joint. The skin over it was hot, œdematous, but not red.

* Read at the Thames Valley Branch of the British Medical Association.

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Temperature 102°. The parents had noticed a slight swelling of the knee for two weeks. After ascertaining that the fluid within the joint was pus by aspiration, I opened the joint freely by a transverse incision through the middle of the patella. The articular surfaces appeared perfectly healthy. With a bent probe I then searched all around the bones at the level of the epiphysial disc or line, and just within the reflection of the capsule, for a sinus or patch of softened bone. Ultimately my probe entered a small opening leading down to a patch of osteo-myelitis on the posterior surface of the femur. This was gouged out and scraped. The result in this case was excellent, and will be referred to under the heading of treatment.

Case 2.—A second case, William R—, æt. 6 months, was admitted the same week. The pus had burst through the upper reflection of the synovial membrane and tracked up under the quadriceps femoris as high as the groin. In this case also the articular surfaces appeared healthy, and it was only after a careful search that a patch of osteo-myelitis was found in the upper end of the tibia, on its posterior surface and about the level of the epiphysial disc.

The symptoms may be more acute than in the above cases, the disease proving fatal in three or four days.

In my experience the knee is more often affected than any other joint, the hip-joint next in frequency,—although by some it is placed first; then the shoulders. Not infrequently more than one joint is involved. The case first quoted above went out well, but returned soon afterwards with epiphysitis near one ankle.

The primary lesion is an *osteo-myelitis*, situated in the growing bone at the extremity of the diaphysis, and in close proximity to the epiphysial disc. Hence separation of the epiphysis from the shaft is not infrequent (see Figs. 1 and 3), especially when the head of the femur is involved. The epiphysis of the head is then found loose in the joint. Whatever joint is affected, the osseous nucleus of the epiphysis itself is rarely involved in the original lesion. The inflammation of the bone

is set up by pyogenic micro-organisms floating in the circulation, which find a nidus suitable for their development in the unstable tissue of the growing bone. They are probably deposited in the newly-formed vessels by a process of thrombosis or embolism. A condition of diminished local resistance may have been determined by a blow or sprain or by exposure to cold. Such lesions not infrequently occur after one of the exanthemata. A scarlet fever, measles, typhoid fever, or diphtheria lowers the general resisting power of the tissues, and the ulcerative and catarrhal affections associated with them open the door to micro-organisms, which are disseminated by the circulation. Cases of arthritis following the acute fevers are sometimes spoken of as scarlatinal, typhoid, or variolar.

The primary osteo-myelitis frequently gives rise in infants to an *abscess* occupying the epiphyseal line. This usually opens near the margin of the articular cartilage; but in rare instances perforates the cartilage near the centre of the articular surface.* In other instances, after death nothing is found except a patch of hyperæmic, softened, and infiltrated osseous tissue at the extremity of the diaphysis, with loosening of the epiphysis.

In other cases, again, the same septic process gives rise to a limited patch of *necrosis* involving the extremity of the diaphysis, the epiphyseal disc, and occasionally a portion of the epiphysis. The joint is usually implicated. The site of the necrosis is well shown in the accompanying woodcuts (Figs. 1 to 3). They are taken from a paper which I published on the subject in the 'St. Bartholomew's Hospital Reports'† for 1879. In it I pointed out the special liability of the growing tissue of bones to inflammation, and referred this to "the active nutritive changes inherent to growth and development of the bone, the multiplication of cartilage cells, development of blood-vessels and hyperæmia at the line of growth, &c." (p. 155).

A precisely similar lesion in the neighbourhood of the epiphyseal line may in older children and young adults give rise to the condition known as *acute necrosis or diffuse periostitis*. The septic products make their way between the periosteum

and the bone, the former being stripped off for a longer or shorter distance. The separation of the epiphysis, so commonly found, points to the site of the osseous lesion as being located in the



Fig. 1.—Necrosis of extremity of diaphysis of humerus and separation of epiphysis. From a young child.

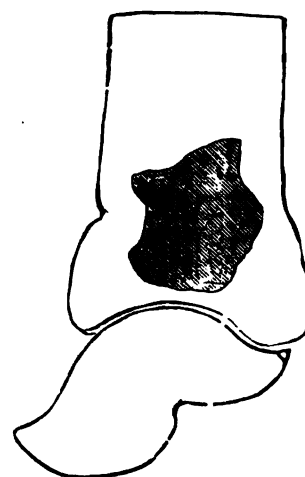


Fig. 2.—Necrosis of extremity of diaphysis and of epiphysis of lower end of tibia. The drawing shows the cavity from which a sequestrum has been removed.

extremity of the diaphysis. In many instances the joint is involved also.

Fig. 3 illustrates all these conditions. The periosteum has been stripped up from the posterior surface of the femur, and a portion of the compact layer (clearly defined in the wood-cut) is necrosed. The epiphysis had separated, and the joint had suppurated.

The fact should not be lost sight of that an abscess starting in an epiphysitis may track up the limb along the surface of the bone, and

* Sir T. Smith, in his original article on "Acute Arthritis of Infants," figures an example of this condition (fig. 5). 'St. Bart.'s Hospital Reports,' vol. x, 1874.

† "On Necrosis at the Extremity of the Diaphysis and in the Epiphysis of Growing Bones."

apparently beneath the periosteum, while in reality it is superficial to that structure. A child, aged one year, was admitted last March with abscesses tracking up the leg due to an osteo-myelitis at the posterior surface of the epiphysial line of the

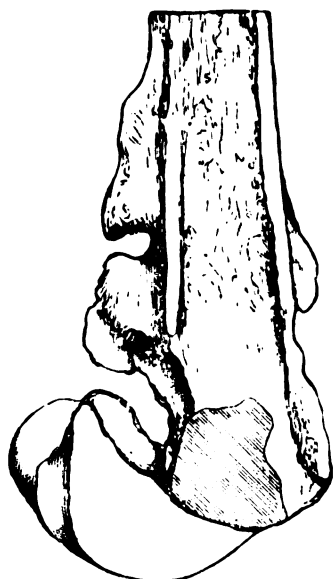


Fig. 3.—Epiphysitis followed by necrosis of extremity of diaphysis of femur (necrosis shaded); displacement of epiphysis backwards; suppuration of knee-joint, and necrosis of portion of compact layer on posterior surface of shaft. From a boy aged 11 years.

tibia. This was scraped, and the abscess drained. Both the tibia and the fibula appeared bare. But after death—which took place six weeks subsequently from pyæmia—the periosteum over both bones was found to be intact.

Acute tubercular arthritis.—Acute arthritis not differing essentially in its clinical characters from the osteo-myelitic or septic group may be tubercular in origin. This affection in the early stage cannot in many instances be distinguished from acute septic arthritis. The onset is equally sudden, and the child is usually brought with the knee or hip distended with fluid. This, on aspiration, is found to be either pus or sero-purulent fluid. The following case illustrates this fact.

Amelia H—, æt. 10 months, was admitted to the hospital with acute arthritis of the right knee. The joint became swollen after a blow against a chair. Temperature 101° . The joint was freely opened, and after some investigation a small opening was found on the posterior surface of the

head of the tibia leading into a sinus which was directed forwards and outwards at the level of the epiphysial disc. The cartilage over the sinus was gouged away, and the patch of softened bone beneath erased. The patient's temperature fell to normal, and the progress of the case was fairly good, but the discharge from the joint had not entirely ceased three weeks after the operation. At that time vomiting with diarrhœa came on, the patient passed into a lethargic state, and died with symptoms of meningitis on the twenty-sixth day after admission. The autopsy revealed tubercular broncho-pneumonia of both lungs, distension of the left lateral ventricles of the brain, with a few tubercles in the choroid plexus. There was considerable erosion of both articular surfaces of the knee-joints, especially of the head of the tibia. The site of the sinus was covered with soft granulation tissue.

In this case it is quite clear that a general tuberculosis was established when the child was admitted to the hospital, and that the osseous lesion was tubercular or the result of a mixed infection with tubercular and pyogenic organisms.

In the above case the *tubercular character* of the affection was only ascertained post-mortem. In



Fig. 4.—Tubercular epiphysitis followed by necrosis in head of tibia. The synovial membrane of knee-joint was pulpy, and there was suppuration. The cavity containing the necrosis did not communicate with the joint. From a boy aged 2½ years.

other cases it may be revealed by the fact that under proper treatment the condition of the joint does not improve. The discharge continues, and ultimately it becomes apparent that there is a distinct tubercular thickening of the synovial membrane. This is confirmed when the joint is laid

open for the purpose of performing arthrectomy or excision.

To indicate still further the close clinical similarity between the tubercular and septic forms of arthritis I may refer to Fig. 4. This represents a section of the head of the tibia, with a necrosis at the situation of the epiphysial line surrounded by gelatinous granulation tissue. No communication existed between the cavity containing the sequestrum and that of the joint. The patient was a boy aged two and a half, who had had inflammation of the knee-joint for no longer than a fortnight. The left knee was hot and tender, the synovial membrane pulpy, and its cavity was distended with purulent fluid.

While dwelling upon the fact that in some instances an acute tubercular arthritis cannot at first be distinguished from a septic one, I must not forget to mention that a joint which has followed the chronic course of tubercular disease may suddenly be attacked with purulent arthritis; this is often owing to the bursting into the joint of a tubercular focus on one of the articular surfaces.

Suppurative syphilitic arthritis.—The well-known affection of the extremity of the diaphysis of the long bones of infants in congenital syphilis, although not infrequently causing loosening or separation of the epiphysis, rarely gives rise to suppuration and disorganisation of the joint. This was, however, observed in the following case. A marasmic infant, with the usual evidences of congenital syphilis, was brought to me at the London Hospital with one of its elbow-joints distended with pus. An incision showed that the articular surfaces were extensively softened and eroded, the whole joint being disorganised. The child ultimately died of exhaustion.

Diagnosis.—The diagnosis of acute arthritis, from whatever cause arising, is usually only too obvious. But in young infants where there is general swelling and œdema, for example of the thigh, and pus has tracked up beneath the muscles from the knee-joint, I have occasionally seen good observers misled owing to the distended joint having discharged its contents beneath the muscles. In infants early diagnosis of the osseous lesion, before the joint has become involved, is probably rarely possible; but this should not be the case in epiphysitis of older children. I think that many cases must be over-

looked in the early stage, because the limbs are not carefully and systematically examined. A child has a slight feverish attack and complains of vague pains in a limb with possibly slight effusion into one or more articulations, which is ascribed to rheumatism. A careful examination would reveal in some instances slight thickening of the extremity of one of the long bones with some tenderness on pressure, perhaps associated with some articular effusion. Should the symptoms not subside there should be no hesitation in cutting down upon and exploring the affected epiphysis. A timely and intelligent intervention in such cases would, I feel sure, not only save many limbs from impairment and mutilation, but also many lives,—for the mortality of acute arthritis and of epiphysitis even with modern treatment is appalling. The disease is from the first of a pyæmic nature, and many patients die with pyæmia or with pyæmic purulent inflammations of serous membranes, whilst others succumb to suppuration and exhaustion. The fatality is, as might be expected, especially high in the case of infants. With a knowledge of the progress made in other departments of surgery by exploratory operations, we should not hesitate to employ them in articular affections.

Treatment.—This is practically that of abscess. The joints should be freely opened, drained, and supported with appropriate splints. In many young children with acute arthritis of the knee-joint I find the gallows splint, of which an illustration is appended, extremely useful. The legs



being raised to a vertical position, there is no risk of contaminating the dressings with the excretions. Continuous irrigation, which I have sometimes employed for the knee-joint, I have scarcely found

practicable at an early age. In one instance in which this was used the child died on the sixth day of septicæmia with pericarditis. The plan of treatment I recommend for the knee-joint, and which I have carried out in these cases, is to open the joint by a transverse incision passing over the middle of the patella, which is divided. The articulating surfaces are thus freely exposed, and an opportunity is gained of ascertaining the situation of the osseous lesion. This should be thoroughly exposed, scraped, and finally swabbed with strong carbolic acid. In this way the suppurative process in the medullary tissue which might ultimately give rise to pyæmia may be arrested, and it is possible to render the joint aseptic. The joint is then flushed, the margin of the patella united by silk or silver sutures, and a drainage-tube inserted. In Case 1 which was treated in this way, the temperature, which was 102° at the time of the operation, almost immediately fell to the normal, and there was extremely little discharge from the drainage-tube, although this was left in as a precaution for some little time. The wound healed by first intention. The patient subsequently left the hospital with a good and moveable joint. The same fortunate results followed the treatment of Case 2, although here the progress was somewhat tedious owing to the existence of a large abscess tracking up the thigh. In dealing with the hip, the joint should be opened up by an anterior incision, and the bone lesion searched for. An opening for drainage should be made on the posterior surface of the joint.

In such operations, and in excisions, I employ a modification of the anterior incision, slight in itself, but greatly improving the view obtained of the articular surfaces. With the patient recumbent, the incision is commenced about one inch vertically below the anterior superior spine, and is carried upwards with an inclination towards the feet, in a curved manner, through the anterior margins of the gluteus medius and minimus, and the tensor vaginæ femoris, till it enters the space between the latter muscle and the sartorius, when it is continued along this interspace in the usual way. This may appear a slight detail, but I insist in dealing with acute arthritis, of no matter what joint, on the importance of employing incisions which freely expose the articular surfaces.

ANTRECTOMY AS A TREATMENT FOR CHRONIC PURULENT OTITIS MEDIA.

BY

W. ARBUTHNOT LANE, M.S.

As several years have elapsed since I first suggested and performed the operation which I called *antrectomy* for cases of chronic purulent middle-ear disease which did not yield to ordinary treatment, I feel that I should now make a general statement as to the subsequent career of these cases after operation, and as to the permanence of the very remarkable improvement in hearing capacity which followed within a very few days upon this treatment, and which was in many cases so complete as to defy the detection of any auditory imperfections by the lay observer.

By antrectomy I mean the obliteration of the antrum completely, or if that is impossible, owing to its having acquired considerable dimensions, sufficiently completely to prevent the formation of a cavity or sinus in the same position.

The details of this operation differ from those adopted by Schwartze of Halle, the pioneer in this section of aural surgery, who exposed the antrum through an aperture in the mastoid process, the diameter of which did not measure more than 15 mm. across. From his description it seems to me that he merely opened and cleared out the antral cavity, and did not of necessity permanently obliterate it, performing an operation of antrotomy rather than antrectomy. Practically, however, antrectomy, though it is a considerable step in advance of his methods of procedure, is but an extended application of the principle involved in Schwartze's operation, to whom we are so greatly indebted for an immense advance in aural surgery.

I would briefly remind the reader of the several points in reference to this operation to which I have directed attention in previous papers, and some of which I believe were more or less original.

I attempted to demonstrate—

1. That the antrum plays a very much more important part in the pathology and causation of chronic purulent otitis media than is generally recognised. It steadily increases in size, and

becomes a progressively increasing obstacle to cure by simple methods.

2. That this cavity had only recently received from surgeons, anatomists, or physiologists the attention it deserves, being ignored by or unknown to some, while by others the term was applied to irregular cavities, the mastoid cells in the substance of the mastoid portion of the temporal bone. In such a comparatively recent and elaborate work as Gruber's text-book on diseases of the ear, translated and edited by Messrs. Law and Jewell, the antrum is figured and described as the orifice by means of which the mastoid cells communicate with the back part of the tympanum. No other allusion is made to the structure in the anatomical portion of this work, nor can I find any description of the manner in which the mastoid cells establish, during their development, this supposed communication with the middle ear.

3. That the antrum has no anatomical or physiological relationship with the mastoid process or its cells, but that it is situated in the petrous bone, and is physiologically and anatomically a part of the middle ear.

4. That the chief if not the sole function of the antrum is to secrete and store up mucus with which to lubricate and moisten the middle ear and its contents, in a manner analogous to the purposes served by the sacculus in the larynx, the surplus finding an exit into the pharynx through the Eustachian tube. It varies somewhat in size in different subjects, but, as a rule, it measures about a quarter of an inch in length by an eighth of an inch in depth and breadth.

5. That a considerable number of mastoid processes consist throughout of very dense bone, or of bone containing but a few minute cancelli.

6. That a large number contain only in the apex of the process, and in its vicinity, cavities which vary in size from minute cancelli to cells of considerable size.

7. That in only a comparatively small number does the mastoid bone contain the two groups of large spaces or cells described by anatomists. It has been pointed out by other observers besides myself, that the older ideas as to the mastoid process being made up largely of cells were absolutely fallacious; and that the presence of dense bone free from any but the smallest cancelli is not the result of chronic inflammatory changes—a mistake

I have known made very frequently by surgeons. What I think is an anatomical or surgical fact of great importance is, that with chronic purulent otitis unassociated with attacks of tenderness and inflammation of the mastoid process (but accompanied by deep-seated pain in the same side of the head and perhaps also deep in the ear), in fact, just such cases as I am calling attention to in this paper,—you are sure or almost sure to find the mastoid process nearly free from cells, and probably also from cancelli; indeed, it is usually composed throughout of very hard dense bone.

8. That in the healthy subject the normal antrum may become continuous with the cells in the mastoid process, owing to their encroaching upon it in their subsequent development. This occasional occurrence accounts probably for the incorrect description of the anatomists.

9. That when a chronic purulent otitis media has existed for some time, the antrum has increased in size, its cavity being filled with decomposing and irritating caseating secretion, which by its presence causes the progressive absorption and destruction of the wall of the cavity. In this way the dura mater of either fossa, with the lateral sinus, may be exposed, and form a portion of the boundary of the enlarged antral cavity.

10. That the antrum, as it increases its area, encroaches more or less upon the mastoid bone, and may establish communication with the mastoid cells if they exist in a high degree of development, these also becoming filled with a material similar to that which is present in the antrum. If, however, the antrum before it had become inflamed were in communication with mastoid cells, the latter of course would participate simultaneously in the inflammatory process.

11. That if the antral cavity be much enlarged, it is perfectly hopeless to attempt, with safety, to clear it of its foul, tenacious, thick contents by any process of irrigation or operation through the external auditory meatus; and even if it were possible to do this it would be only a matter of a few days or weeks before the antrum had again become choked with decomposing secretion.

12. That while the enlarged antrum is filled with its irritating contents, aerial conduction may be partly or completely absent, and that within a few days after the operation of antrectomy and cleansing the middle ear, though the patient may

have been absolutely deaf in that ear for more than twenty years, ordinary conversation will be heard readily. In such a condition it is just a little difficult to understand by what means the presence of the caseating material in the middle ear and antrum is able to keep the hearing capacity of the internal ear in abeyance, so that within a few days after its removal the acuteness of hearing becomes as keen as ever. It is probable that it acts partly mechanically as a buffer to sounds, and partly by keeping up inflammatory irritation, much as the dry crusts sometimes do in ozæna.

13. That the facial nerve bears a very important relationship to the aperture by means of which the antrum communicates with the middle ear, since it lies immediately to its inner side, and that any uncertain interference with this aperture may result in permanent damage to the nerve.

14. That the chief function of the membrana tympani is to prevent evaporation of the secretions of the middle ear and antrum, and that its presence is not necessary in order to hear ordinary conversation with acuteness.

15. That after antrectomy, in order to retain the hearing capacity in the improved condition resulting from the operation, it is necessary to replace the functions of both the antrum and the membrana tympani. The former is done by daily irrigation, and by the introduction of a minute quantity of some antiseptic, as boro-glyceride or a mixture of glycerine and iodoform, and evaporation is prevented by placing in the meatus a small plug of lamb's wool.

I have now performed antrectomy a very considerable number of times upon patients suffering from chronic purulent otitis media, and very often from pain, either more or less continuously, or at intervals, over the whole side of the head, or perhaps only over the mastoid process and above the ear; sometimes neuralgic in character, and at other times described as being boring, or throbbing, or like a deep-seated abscess, and very often preventing the patient from lying on that side of the head when in bed. In many of these cases the patient did not complain of pain or tenderness when the mastoid process was forcibly compressed with the thumb, or struck with a pleximeter. Such patients not uncommonly have a slight but distinct rise of temperature in the late afternoon or evening, showing that some absorption of septic

products takes place constantly from the antrum, and produces a harmful influence on the health of the sufferer.

I need hardly point out to you how great a source of danger to the patient is this cavity filled with many varieties of organisms, which are capable of setting up mischief elsewhere, when the vitality of the individual is depreciated generally or locally. It seems to me that the profession generally are hardly sufficiently alive to the importance of this, and do not regard the diseased ear with the fear and suspicion it merits.

In all, or nearly all these cases, the antrum was obviously very definitely enlarged—in most cases very considerably so,—and, what is a matter of the greatest importance, the mastoid bone hardly ever contained any but the most minute cancellous spaces; but, on the other hand, it was often so dense as to take a good hour's hard work or more, with gouges and a heavy mallet, to expose the antral cavity.

I am not, of course, considering the conditions that are so common in inflammation of and suppuration about the mastoid process. One has heard of mastoid cells distended with pus being readily opened by the use of even so clumsy and unsuitable an instrument as the trephine, and then being cleared out with a sharp spoon; possibly, too, the antrum is also found and cleansed, especially if much enlarged and communicating with the cells by an aperture of any size.

I do not suppose that any surgeon in the present day would be sufficiently unfamiliar with the anatomy of the part as to venture to apply such a dangerous implement as a trephine for the purpose of exposing the antrum; but it is not so many years ago since it was recommended and made use of, not unfrequently with disastrous results.

It is at once obvious that the deep-seated and enlarged antrum, covered almost always, in the cases I particularly wish to call attention to, by about three quarters of an inch or more of very dense bone, is infinitely more dangerous to the life of the individual than an antrum in immediate relationship, or in direct communication with large cells in the mastoid bone; since the latter, when inflamed, at once makes itself obvious to the most inexperienced surgeon, and even if he does not interfere, the pus usually escapes externally at last.

That the pain in the head from which the patient with the enlarged antrum suffers is in most cases due to an obvious chronic inflammation of the dura mater in immediate relation with the antrum, I have been able to demonstrate on very many occasions.

By exposing the antrum in such cases, by the careful use of the mallet and gouges, and by scraping the cavity with sharp spoons, and then by subsequent removal of overhanging bone, so as to make the gouged inner wall of the antrum the floor or apex of a cone, the base of which is rendered as broad as possible in order to remove the sides of the enlarged antral cavity, and by plugging the cavity for a considerable time with iodoform or other gauze, till the skin forms a dimple over the obliterated inner wall of the antrum, its cavity is permanently obliterated, and the floor of the cone becomes partially filled up with fibrous tissues. The duration of the operation in the case of very dense bone may be much curtailed, and the operation itself facilitated by the use of an electric motor and burrs. If the middle ear is much diseased it is then thoroughly cleared of its contents, any relic of the membrana tympani being carefully removed, and the aperture of communication with the antrum may be somewhat enlarged by the removal of portions of its outer boundary, the whole of the posterior boundary of the external auditory meatus having been left intact as far as possible when the mastoid process was cut away. In performing this last stage of the operation the greatest care must be taken to avoid any damage to the facial nerve, and up to the present I have succeeded in doing this. On one occasion the muscles of the side of the face twitched violently, but no damage resulted. I may say that though it has never been my misfortune to damage this structure, it is, I believe, the chief—I may say the only—risk of the operation; yet it is one that would be most distressing to the patient, and should therefore always be in the mind of the surgeon should he consider it advisable to enlarge the aperture into the middle ear.

If there is no perforation in the membrana tympani, or if with a perforation of small size there is no evidence of extensive disease of the middle ear, the membrane may be left untouched with advantage.

As regards the subsequent condition of the

patient, months or years after the operation, when the simple daily routine of cleaning the ear and introducing a plug into the meatus is followed, the hearing capacity not only retains its improved condition, but often becomes more acute. If, however, the patient is dirty and careless, and pays no attention to the ear after he passes from observation, the hearing capacity gradually diminishes owing to the accumulation of dry secretions and inflammatory materials in the middle ear. My experience of the operation is that it is one of the most satisfactory and useful operations that we have in surgery. In skilful hands it is accompanied by practically no risk; it is followed by no pain worth talking of; it absolutely frees the patient from subsequent risk from intra-cranial complications if the simple directions as to cleansing, &c., are followed; it removes the foul discharge; the headache, neuralgic pain, and tenderness disappear; it very often cures any existing facial paralysis; it prevents the formation of aural polypi; and it gives the patient back almost perfect hearing, which remains in the same condition or improves if he takes with it only such small trouble as the ordinary cleanly person habitually devotes to his teeth.

I would point out, however, that by ventilating the naso-pharynx systematically and habitually, and in this manner diminishing largely the tendency to obstruction of the Eustachian tube, it is possible in many cases to relieve the patient sufficiently of the inflammation of the middle ear and antrum as to obviate the necessity of performing antrectomy. I have also found these simple means of the greatest service in other conditions of the ear due to Eustachian obstruction.

DR. S. SOLIS-COHEN frequently prescribes the *fluid extract of quebracho* in the treatment of asthma, whether of the bronchial or purely spasmodic variety. The dose is from 30 minims to a fluid drachm, and may be repeated hourly or less frequently, according to circumstances. When the stomach is irritable it should be given in a bland vehicle, or some liquid preparation of pepsin. It acts promptly or not at all. Unless relief is experienced within forty-eight hours it should be discontinued. When useful it may be continued in combination with a sedative.—*Philadelphia Poly-clinic*, October 2nd, 1897.

CLINICAL LECTURES ON URINE.

Delivered at University College Hospital by
J. ROSE BRADFORD, M.D., F.R.S., F.R.C.P.,
 Physician to University College Hospital.

IX.

THE phosphates are the salts of the urine which are of most importance clinically, and the phosphates in the urine (we must repeat what we have said about the sulphates and chlorides) are mainly from the phosphatic material in the food, and partly from the tissues in the body; but many persons with a smattering of chemistry think that the phosphates of the urine are derived mainly from the tissues of the body. The amount of a phosphatic deposit in the urine is no index to the amount of phosphates actually present. The phosphates present in the urine may be divided into two groups; there are the earthy phosphates and the soluble phosphates. Phosphate of soda is the typical representative of the one, and phosphate of lime is the typical representative of the other; phosphates of magnesium and phosphates of soda represent the soluble form, and phosphate of lime represents the insoluble. It is useless to make any observations on the amount of phosphates in the urine, except with the uranium nitrate method, which estimates all the phosphates present.

There are two points to be considered about the phosphates, firstly the amount of phosphates in the urine, and secondly the mode of their excretion, and in this respect the phosphates resemble the urates. When we were doing the urates, you will remember I said that the mere amount of uric acid is often of secondary importance. The main point is often the condition determining its deposition, and the percentage amount of uric acid in the urine is only one determining factor. So it is as regards the phosphates; the question of practical importance is what are the conditions which lead to the deposition of the phosphates in the urinary channels, and in these conditions very often the amount of phosphate in the urine is the least important, so that the long and the short of the whole matter is, that the quantitative determination of phosphates in the urine is but rarely done. The thing to study is, what are the conditions

which lead to the deposition of phosphatic material in the urine.

The solubility of the salts of phosphoric acid, particularly of the lime salts, depends on the nature of the salts, whether mono- or di- or tricalcic salts—the tricalcic salts being very insoluble, the dicalcic salts being more soluble. The question of the solubility of the phosphates in the urine is very largely a question of the reaction of the urine, and you will remember that urine if it is alkaline may be made alkaline owing to the presence of volatile alkalies or owing to the presence of fixed alkalies. Every one knows that decomposing urine becomes muddy owing to the precipitation of phosphates, and the phosphates that are thrown down under these circumstances are the triple phosphates, that is to say the ammonio-magnesium phosphates; that is a condition which occurs in urines exposed to the atmosphere, and undergoing decomposition. That may occur in the pelvis of the kidney as the result of pyelitis, or in the bladder as the result of passing a dirty catheter. If an organism that decomposes the urine is introduced, you will have ammonio-magnesium phosphate formed; it is a crystalline deposit, and it has a great tendency to form a stone, but rarely in the kidney, more frequently in the bladder, and here the stone is usually formed by phosphatic deposition around a nucleus of some other material, *e.g.* uric acid. The fixed alkali will lead to the precipitation of the earthy phosphates in an amorphous form; this condition we all of us have at times, if you watch your urine you may observe it two or three hours after meals. There are very few people who do not occasionally pass milky urine from the precipitation of phosphatic substances. Nervous individuals notice this milkiness, and consider it due to the passage of seminal fluid, or else think it due to phosphatic material derived from the metabolism in the body; they get into the hands of unscrupulous persons, and they may become very ill from hypochondriasis and other allied ailments. It is a condition that is seen in very slight departures from health, more particularly in cases of slight indigestion, and that is a condition you understand, that is not in any way due to an excess of phosphatic material in the urine; it is due to diminution in the acidity of the urine, leading to a precipitation. If you succeed in making the urine acid, the milkiness disappears.

As to how the urine is best made acid the way is to alter the diet, order a diet that is relatively poor in vegetable matter; anybody who eats largely of vegetable matter is bound to get an alkaline urine with this phosphatic deposit. You should give benzoic acid or some compound such as ammonium benzoate, which is a fairly certain method of increasing the acidity of the urine; they are both very nasty drugs to take, hence they are usually prescribed in cachets. You should not try and increase the acidity by giving mineral acids, for mineral acids in large quantities tend to damage the kidney. There are several other phosphates to be considered; there is a phosphate of lime that is known as a stellar phosphate, because it crystallises in long needles, and forms rosettes, something like the rosettes of tyrosin crystals, but they are not coloured like tyrosin tufts, which are greenish, whereas the phosphatic ones are not; they are also usually bigger. I cannot tell you why these crystals form, that is not known; it is probable that the same explanation holds that holds in the case of the oxalates. You occasionally get this crystalline stellar phosphate formed in more or less normal urines, and they may give rise to trouble by the formation of a calculus. But a phosphatic calculus is almost always secondary; it is formed in the following way generally. There is a uric acid calculus formed in the kidney, and coming down into the bladder cystitis is set up, decomposition of the urine occurs and you have a triple phosphate deposit outside the uric acid nucleus.

There is one more point about the phosphates, viz. the confusion with albumen. When you warm urine containing only a small amount of acid and *a fortiori* if alkaline, a cloud appears, and that cloud is a calcic phosphate; there has been a great controversy as to the explanation of this precipitate. There are the following views held with reference to this question; two of them are certainly rather doubtful, but one is obliged to mention them. It has been said that the boiling of this urine which is deficient in acid leads to the precipitation of phosphatic material, owing to the formation of ammonia from the urea. If you boil a urine that is very nearly alkaline, you decompose the urea into ammonia, and the increase in the alkalinity by the ammonia is said to be sufficient to precipitate the phosphate. When you boil such a urine you decompose some urea—Shattock showed that,

—but you have to boil it for some time, and this deposition of phosphates occurs long before the urine is boiled, it occurs as soon as the urine is warmed; and therefore this explanation cannot, I think, be accepted, first, because it requires more or less prolonged boiling to decompose the urea, and secondly because it occurs without boiling. The second explanation was that the phosphates were kept in solution by the carbonic acid, and that on boiling, the carbonic acid was driven off, and therefore the phosphates came down; but the same thing occurs if the urine is boiled in a sealed tube. The most likely explanation is that the deposition of the phosphates on warming is owing to an interaction between the phosphates, forming the tricalcic phosphate; the cold urine contains dicalcic phosphates, and as a result of warming, two molecules of dicalcic phosphate become converted into a molecule of monocalcic phosphate and a molecule of tricalcic phosphate, and the tricalcic phosphate is insoluble, and comes down. The cloudiness appears on warming, and on being cooled again it disappears, if the test is done in a sealed tube.

We will now pass on to the oxalates. Oxalates are important, owing to the formation of calculi, the oxalate of lime calculus. The mulberry calculus is not very common; but it is a very serious form of stone, owing to its hardness, and it has led to accidents to instruments in the attempt to crush it. Oxalate of lime is also important, because you can have little minute microscopic stones causing hæmaturia. Some elderly patients will have hæmaturia, and there is little doubt that it is due to small oxalate stones. The oxalates are present in small amounts in normal urine, but certain persons apparently make more oxalic acid than others; there is apparently an oxalic acid diathesis, as we have seen that in all probability there is an uric acid diathesis. In diabetes you get an excessive amount of phosphates excreted, and one French observer holds the view that phosphaturia always precedes glycosuria.

Some physicians hold that persons suffering from oxaluria are dyspeptic. If you examine the urine of hypochondriacal patients, you may find traces of oxalates. The most important condition in which you have oxalates in the urine is undoubtedly as a result of eating vegetables, more particularly of coarse rhubarb. You would be surprised

at the great number of persons who get rather severe symptoms from this. I have seen several cases of patients who have had severe symptoms—pain in the back and hæmaturia—as the result of oxalates; the whole thing has been produced by eating rhubarb and various other vegetables. It follows, therefore, that you ought always to think of a vegetable source for oxalates in the urine. An interesting point has been worked out with reference to the formation of calculi consisting of oxalates; we know most about it in the case of the oxalate of lime calculi. You will remember when doing uric acid I drew your attention to several conditions which tended to cause the precipitation of the uric acid, but there is always the question as to what causes the first crystal; the growth is easy to understand, but what causes the first crystal is more difficult. It has been shown in the case of oxalates that the presence of proteid matter or even of bodies allied to proteids, mucin, influences the crystalline formation very greatly. If you take a solution of oxalic acid, and add some lime to it and get crystals formed, if you do it in an ordinary medium you get minute crystals; but if you do this in a medium with a small amount of albumen or mucin, you get much larger crystals. If you cut sections of the mulberry calculus—and there is a very interesting series of them published in the Pathological Society's 'Transactions,'—you may find that the nucleus of the stone is one big crystal. It has long been known that patients with oxalic acid calculus have it often only on one side; uric acid stones are perhaps more often seen on both sides. It has been always difficult to explain that on the diathesis theory, and these observations to a certain extent do not explain it, but they throw some light on it. If a patient tends to excrete larger quantities of oxalic acid than he ought, if he gets a little pyelitis or hæmorrhage into the pelvis of one kidney, he will run a risk of forming a stone. A small amount of proteid material will influence the crystallisation to such an extent as to make a salt (which usually crystallises in small microscopical crystals that give no trouble) crystallise in such large crystals that one of these may easily act as the nucleus for the subsequent growth of a stone. This has not been shown in the case of uric acid, but a somewhat similar phenomenon is seen with this body, as in crystallising it has an extraordinary

affinity for combination with pigment. It is more or less of the nature of a combination, and to a certain extent this fact resembles those described for oxalic acid, since the pigment not only combines with the uric acid, but it influences also the shape of the crystals. Oxalic acid in the urine crystallises in the form of needles and rhomboids; when pure it is in plates, and you understand that the nucleus, of at any rate oxalic stones, is a single large crystal, and one has definite experimental evidence that such a crystal can be obtained by adding proteid material to urine containing oxalic acid.

CHRONIC PNEUMONIA, CULMINATING IN DESTRUCTION AND EXCAVATION OF THE LUNG.

From the *Australasian Medical Gazette*, August 20th, 1897. By S. H. MACCULLOCH, M.B., CH.M., and ANGEL MONEY, M.D., F.R.C.P.

THE patient was a powerful man of great muscular and osseous development, and weighing over seventeen stone; his age was about 39. He was a bookmaker, and had lived a hard life, with the use of much alcoholic stimulation. He was suddenly taken ill on the 29th March, 1897, with all the symptoms and signs of a typical attack of acute pneumonia, rapidly involving the whole of the right lung, and spreading from the base continuously upwards. The case appeared to be proceeding in a satisfactory manner, but the dulness never cleared, and on the 17th April apparently a relapse set in, or at least there were renewed signs of fever and those disturbances naturally attending the classical form of extensive lobar pneumonia. Both of us saw the patient on the 6th May (*i. e.* thirty-eight days after the initial symptom), when the physical signs indicated complete solidification of the whole of the right lung, with perfect preservation from disease of the left lung. The dulness was so extensive, and so completely followed the outline of the lung, that, notwithstanding the vocal fremitus was everywhere rather increased than diminished, it was deemed advisable on several occasions to insert the exploring syringe. The indications, gained by all methods of examination, proved the absence of serous or purulent pleural exudation. The opinion formed by us was that the whole lung was solid, and that the form of consolidation was that which had been termed by

Addison "albuminised." Addison described a rare class of case, in which, after an illness of a few weeks, with all the evidences of consolidation, the patient dies, and the lung is found uniformly "albuminised." The section is not soft, lacerable, and granular, as in acute grey hepatitis, but smooth, solid, and tough, the exudation having undergone a fibrillary change such as might end in sclerosis. Dr. Hilton Fagge, after quoting the above from Addison, states that this is a condition of lung very liable to break down, and thus constitute a case of rapid phthisis. He goes on to say also that it is a condition from which recovery may take place and the lung be completely restored. Fagge further quotes from Wilks and Moxon:—"That there is a chronic pneumonia of such a kind can scarcely be denied, when it is remembered for how long a time all signs of consolidation may endure, and then a complete restoration take place. We must, therefore, believe that there is a true chronic pneumonia, whose origin is in ordinary inflammation and exudation into the alveoli, and whose appearance is best denoted by the term 'uniform albuminous induration.'" They add, however, that such chronic pneumonia may be of the lobular kind, and Addison himself said that this condition may be limited to one or a few lobules only; so that it may, perhaps, be doubted whether this "least frequent of the permanent pneumonic indurations of the lung" is not, after all, an unusually extensive catarrhal pneumonia.

Certain it is, and this must be well known to those who have much post-mortem experience of cases of broncho-pneumonia, lasting a few weeks, in children, that the consolidation may be so tough and indurated as to resist any attempt to thrust in the thumb-nail. One of us has verified this several times, and the consolidation has been lobar in distribution, though catarrhal in histological characters.

In the case which is under discussion, the patient began on the 22nd May to expectorate large quantities of stuff which resembled nothing so much as anchovy sauce; and this expectoration evidently meant that extensive destructive excavation of the lung was proceeding. Because, on the 16th June, when we again met in consultation, amphoric breathing and a bell-sound could be detected, not only in the upper lobe in front, but in the lower lobe behind and at the side. Further

corroborative evidence of this diagnosis was obtained by operative interference, which it was deemed offered the best chance for the patient's recovery. Dr. MacCormick and one of us made incisions into the lung and drained the cavities in question, the upper and lower vomicæ being easily made to communicate with one another. Unfortunately, two days (June 14th) before operation (June 16th) was decided upon a secondary infection of the right extra-thoracic subcutaneous and intermuscular tissues took place, and a large phlegmonous abscess formed. The signs indicating this were a boggy swelling at the side, and behind on the right side, with the development of renewed fever and profuse perspirations. For many weeks there had been hardly any fever, and the period between April 22nd and June 14th was almost one of apyrexia. At the operation a pint or rather more of offensive pus was let out of this extra-thoracic formation. There was no direct continuity between the excavation in the lung and the abscess outside the ribs.

Following these surgical measures a further gangrenous and emphysematous inflammation spread widely over the whole of the right side, and extended up to the neck and beyond the middle line to the opposite side of the body; but it was entirely outside the bony parietes, and it evidently determined the patient's death as a process of pyæmia.

It was remarkable that the muscular strength of the patient continued scarcely unabated after the subsidence of the original pneumonic pyrexia; and also, on the other hand, that the pulse, though not of greatly increased frequency (84 to the minute), was singularly soft, so as to be easily extinguished by the slightest digital pressure. The main suffering of the patient till the fatal infection set in consisted in the presence of severe pain, which continued throughout, and could only be kept in check by the frequent exhibition of morphia subcutaneously. A necrosis of the seventh and eighth ribs was discovered at the operation.

Although for the sake of description such a termination of wide-spread pneumonia may well be called rapid phthisis, there can be but little doubt that the destructive process was not of a tubercular nature; there were no tubercular bacilli in the anchovy-sauce like expectoration; nor was the process of the kind known as purulent infiltration.

Our belief is that the case was one of that very rare kind in which the original pneumonia went on to the process called chronic interstitial pneumonia. Osler's experience gives but one case, proved post-mortem, of this mode of termination of acute croupous pneumonia; it was that of a man *æt.* 58, who died on the thirty-second day after the initial chill.

We have both considered that alcoholism was the chief *ætiological* factor in determining the unusual sequel in this case. Be that as it may, the fact of marked excess in the use of stimulants was admitted by everyone, and physical examination led us to believe that the liver was sclerosed. How far the heavy weight of the man and his defective blood pressure may have contributed to prevent the ordinary resolution of the pneumonic lung is a matter for consideration. Sir R. D. Powell, in his book on 'Diseases of the Lung,' avers that the too prolonged use of stimulants during convalescence tends to delay absorption; and we think that it may, with previous excessive use of alcohol, lead to sclerosis of the exudation and alveolar walls.

The total duration of the case was eighty-six days, or twelve weeks; and the period of consolidation, prior to excavation, lasted fifty-four days, or nearly eight weeks. The sputa at the beginning were never actually rusty, but prune-juice coloured, although as tenacious as is customary in acute pneumonia. We both regret not being able to obtain a post-mortem examination; but the clinical facts spoke so plainly that there can hardly be any doubt as to the correctness, in the main, of the pathological diagnosis. The operation proved the induration of the lung around the cavities, and the only unsolved point concerning the pulmonary process was whether the exudation was originally croupous or catarrhal; this difficulty has already been mentioned in the quotations from Wilks and Moxon:

TUBERCULIN.

IN No. 16 of the 'Correspondenz Blatt f. schweizer Aerzte' for the current year there appears an article by Dr. Tavel, of Berne, in which he gives a *résumé* of all the work done in immunisation to tuberculosis in general and especially in relation to Koch's new tuberculin. Dr. Tavel does not express any opinion on the therapeutic value of the prepara-

tions noted, but only gives the experimental facts on which the present status of specific therapeutics in tuberculosis is based.

The author distinguishes insusceptibility to bacteria—*i. e.* true immunity—from insensibility to their toxins; the former as bacterial immunity, the latter as toxin immunity. The one is entirely independent of the other; thus, for instance, the immunity produced by Pasteur against anthrax and that by Pfeiffer against typhoid fever and cholera (bacterial immunity) are sometimes followed by an increased susceptibility to the toxins.

In the production of artificial immunity two ways have generally been followed. The formation of the "anti-bodies" is left to the organism to be immunised (the active procedure); or the "anti-bodies" are produced by a foreign organism, that of the horse, for instance, and the serum, containing the immunising properties, subsequently transferred to the organism to be immunised (the passive procedure).

Active and passive immunisation were first attempted by means of the metabolic products of the bacilli. The tuberculine which, in Koch's opinion, represented "a glycerin extract from the pure cultures of the tubercle bacilli," cannot be considered as such. It has to be looked at as an extract of the metabolic products which adhere very closely to the bodies of the bacilli. This peculiarity was well shown by the experiments of Strauss and Gamaleia and by Prudden and Hodenpyl, by intravenous injection of dead tubercle bacilli. These toxins penetrate only slowly into the surrounding tissues, a fact which satisfactorily explains the local process of tuberculosis, the tubercle, and the unimportant *rôle* intoxication plays in this disease.

The author further enumerates the attempts made to produce preparations without the dangerous properties of the crude tuberculin and of higher efficacy. Precipitation with dilute alcohol and subsequent solution gave the *tuberculinum depuratum* of Koch, with an action five times stronger than that of crude tuberculin. Precipitation with bismuth iodide of sodium and alcohol led to the *tuberculocidin* of Klebs, and, with some modification of the chemical procedure, to *antiphthisin*. Tuberculin and tuberculocidin are chemically different from antiphthisin, in the latter the extraction of the tubercle bacilli being entirely avoided.

The development of reactive processes in the treatment of persons with tuberculin has been supposed to be essential. As the products of these reactive processes were obtainable in the organism of animals and transferable by means of the serum, this method was taken up first by Héricourt and Richet as early as 1891. Their experiments were followed by many others, especially those of Nieman, who showed clearly the possibility of producing an antituberculin.

The other and most recent method by which immunization has been attempted is, as the author states, that with the proteids of the tubercle bacilli. Koch, who has published the latest results in this direction, obtained by mechanical destruction of the cellular membrane and extraction and solution of the protoplasm two preparations which he calls TR and TO. Dr. Tavel only recapitulates the facts given by Koch in his last publication on the subject; he regrets that no exact animal experiments were published in order to support the affirmation that TR possesses immunising properties.

The author, finally, believes that the experiments of Viquerat promise good results. They consist in the production of an immunising serum from partially immune animals. The mule shows the peculiarity of responding to an infection with tubercle bacilli (by intravenous injection), but readily overcomes the attack. There is a formation of tubercles and there is proliferation of the bacilli at first, but this soon stops; in a hundred days Viquerat as well as Tavel has not been able to find any more living bacilli. During the process of restoration there is a continuous production of "anti bodies," which circulate in the serum. This serum, injected into human beings, produces a passive bacterial immunity. In conclusion, Dr. Tavel gives a *résumé* of all the different methods of antituberculous specific therapy:

1. Active immunisation with the metabolic products of tubercle bacilli: Tuberculinum Kochii, Tuberculinum depuratum Kochii, Tuberculocidin (TC) Klebs, and Antiphthisin Klebs.

2. Passive immunisation with the serum of animals immunised with tuberculin (tuberculinised): methods of Héricourt, Richet, Maragliano, Nieman, and others.

3. Active immunisation with the proteids of tubercle bacilli: Tuberculin (TR) Koch.

4. Passive immunisation with the serum of animals treated with living tubercle bacilli (tuberculinised): Viquerat,—*New York Medical Journal*, 25th September, 1897.

TOBACCO AMBLYOPIA.

A CHRONIC retro-bulbar neuritis may arise from a variety of causes, chief of which is the abuse of tobacco. From its most common cause the disease has been called "tobacco amblyopia," though it is better to keep the term "retro-bulbar neuritis" as derived from the pathology, rather than to accept a name derived from the ætiology. The victim usually comes early to the physician, complaining of some loss of sight, with possibly a slight cloudiness obscuring vision. In such cases the ophthalmoscope reveals no signs of a special pathological state, and normal vision soon returns on the discontinuance of the use of tobacco.

In advanced cases, vision may range from $\frac{20}{200}$ to $\frac{5}{200}$. Reading is impossible; a hazy cloud is extremely annoying; central vision is so poor that mechanically the patient will turn his head to either side in order to use the peripheral parts of the retina. He is often sure he sees better at night, at least he does not see this haziness quite so disagreeably. This oft-observed nyctalopia caused Arlt, ignorant of the pathology, to call this form of neuritis "retinitis nyctalopica." The patient does not know when the trouble began. He may early detect his inability to read as well as formerly, but many of these patients (who read but little) are not troubled much until failing vision interferes with their work. Then some consult the physician at once; others not realising the trouble lose their positions, and console themselves with their beloved pipes and coddle their cud until a condition of almost complete blindness drives them to some hospital to seek advice.

The patient tells enough to suggest at once the diagnosis. If the ophthalmoscopic examination is negative, the physician examines for central colour scotoma in that part of the field corresponding to the part of the retina supplied by the papillo-macular bundle of the optic nerve. A careful examination will show a colour scotoma in both eyes, with loss of central vision. As the retina is very easily exhausted, especially at the border lines of the scotoma, it is not advisable to try to map

out the boundary lines too carefully. Have the patient look at a black-lead pencil a few feet in front of him, and then quickly present to the nasal field of the eye examined a small black disc with a red centre. At once the patient recognises the colour. When the disc is brought to the temporal side, the patient cannot possibly recognise the colour in the oval field corresponding to the maculo-papillary retinal region, or in mild cases he will at once notice that the colour is less bright. In advanced cases he may not recognise bright blue or even white.

The peripheral parts of the field are colour-blind in the normal eye. The nearer the colour approaches the centre of the field, the more acutely is the colour recognised. In this form of amblyopia, since there is an atrophy of the very nerve-fibres that best distinguish colours, we expect the patient to have central colour scotoma, and should remember this as the leading point for diagnosis in this form of amblyopia.

The ophthalmoscopic examination is usually negative. From a close examination the physician is usually convinced (from the breath) that most of these patients indulge in the abuse of whisky as well as in that of tobacco, so that in many cases it is a question which should take the greater blame. I am convinced from the pathology of the abuse of alcohol (increase of connective tissue, decrease of normal cells) as well as from the breath, that alcohol must be considered as an adjuvant to the tobacco in such cases. Some believe that alcohol alone can produce this retro-bulbar neuritis. Noyes states in his text-book that "in alcoholic amblyopia we find usually a dull red nerve with swollen veins, rather hazy borders, and torpid circulation. Atrophy may subsequently ensue. . . . In tobacco amblyopia the nerve is brighter and more nearly normal, or it may show tokens of atrophy or of interstitial inflammatory exudation. Generally there is little lesion to be recognised."

Since Samelsohn showed that the pathology of this form of amblyopia lies in the maculo-papillary bundle within the optic canal, we look closely at that part of the fundus which is supplied by this bundle of nerve-fibres. We are not surprised to find the temporal side of the nerve whiter than the rest of the nerve, so white in some cases as to indicate atrophy, and to cause surprise that this peculiarity has not been more particularly mentioned.

The working man living out of doors, and consuming tremendous quantities of food and oxygen as well as tobacco, is usually the victim. The cigarette smoker escapes because his stomach gives out before his optic nerve is in the least danger. Were the "gentleman" smoker to consume the quality and the quantity of tobacco that some of our working men smoke with keen pleasure, the result would be an attack of acute nicotine poisoning.

I personally am very fond of tobacco, but a few whiffs from a hod-carrier's vade-mecum would satisfy my tobacco craving, at least until after dinner. There is more nicotine-paralysing ability in the smoke from one pipeful of black "juicy" tobacco than in a hundred mild cigarettes, yet occasionally we hear of mild amblyopia from cigarette smoking. Were the working man to use milder tobacco and to clean out his pipe occasionally, this form of amblyopia would rarely be seen; but so long as the strongest kinds of tobacco are sought for and enjoyed, so long will tobacco amblyopia be encountered.—RICHARD ELLIS, M.D., *Medical Record*, Sept. 25th, 1897.

EVIDENCE WHICH TENDS TO WEAKEN THE VALUE OF WIDAL'S TYPHOID AGGLUTINATION REACTION.

To one who listened to the recent elaborate and very valuable discussion upon sero-diagnosis in typhoid fever at the last meeting of the Association, the testimony upon the value of this test seemed far from convincing. The discussion was carried on by some of the most prominent bacteriologists in America, and dwelt in considerable detail upon many features of the Widal reaction. Still there was a diversity of opinion upon many essential features; and even after the report of the special committee appointed by the chairman to harmonise the views expressed, there were still elements of uncertainty about the matter.

A great deal was said about the importance of the technique to be employed in making the Widal serum test, and still there was scarcely a single point in the methods in which two observers agreed. Opinion differed as to the source of the serum, whether it should be from blood, from blisters, or whether dried blood should be employed; though the reaction was obtained by

serum from any of these sources. The dilution of the serum was another point upon which opinions varied, and this held true for the source, age, and virulence of the typhoid culture; for the dilution of the bacterial emulsion, and for the proportion of emulsion and serum. Then came the question of the agglutination reaction. What should be considered a reaction? How far should loss of motility and agglutination proper be given value? What should be considered the time limit of a reaction?

The single point upon which an almost unanimous opinion was obtained was with reference to the clinical value of the Widal reaction as a sign of typhoid fever.

The diversity of views expressed by these representative American scientists is only a repetition of the opinions of observers in all parts of the world who have discussed this question; and while these discussions, especially when conducted as systematically as the one at Philadelphia, will soon enable us to decide upon the merits of the case, they all seem to foreshadow a disappointment for those enthusiasts who failed to exercise a due conservatism in forming their judgment upon the value of the Widal test. Even from the clinical stand-point there can scarcely be a doubt that the reaction has occasionally failed to appear when the blood of a typhoid fever patient has been used; and, on the other hand, the reaction has occasionally been obtained in a variety of other diseases. How far these results have been due to faulty technique is impossible to say, especially when it is so hard to decide upon what is to be regarded as *the correct technique*.

It is well known among bacteriologists that there is a close biologic relationship between the colon bacillus and the bacillus of typhoid fever; so close, indeed, is this relationship that many bacteriologists have come to regard the common bacillus of the intestine and the bacillus of typhoid fever as varieties of the same species. This view has been advanced from time to time, and is again strongly suggested in the elaborate studies by Adelaide Peckham ('The Journal of Experimental Medicine,' vol. ii, No. 5, 1897). Among other interesting and valuable studies upon these bacilli, this author has tested a large number of colon and typhoid bacilli from various sources as to their reaction with typhoid blood serum. A

considerable number of cultures of what were regarded as typical typhoid bacilli failed to give a characteristic reaction, while at least two out of nineteen typical cultures of *B. coli* responded perfectly to the serum test. These results are quite in accord with all the other so-called specific methods for the differentiation of the typhoid and colon group of bacteria, and they would seem to be a serious blow to the Widal tests, for upon the specificity of the reaction hinges much of its value in clinical diagnosis. If the colon bacillus will respond to the agglutination effect of serum from typhoid patients, why will not the typhoid bacillus be agglutinated by the blood serum from colon bacillus infections? We believe this will be found to be the case, and in support of this opinion we have succeeded in obtaining a typical agglutination reaction with typhoid cultures in five out of seven cases of suppurative appendicitis by using the dried blood after the directions of Johnston. In three out of the five positive cases a bacillus corresponding perfectly to the colon type was alone isolated from the pus obtained at operation. The other cases had already been operated upon, and no analysis of the pus was made. Such results seem suggestive, to say the least, and they should be followed up.

Another curious feature about the agglutination of the typhoid bacillus has just been brought out by the experiments of Malvoz ('Annales de l'Institut Pasteur,' t. xl, No. 7, 1897), who has succeeded in producing a reaction with typhoid bacilli, similar to that produced by typhoid serum, by various chemic substances. He mentions especially formaldehyde, corrosive sublimate, peroxide of hydrogen, and strong alcohol. Among the aniline colours, crysoidin, vesuvin, and safranin have the property of provoking a perfect agglutination even in very dilute solutions. Malvoz attempts to use this agglutinative action of chemicals for the differentiation of colon and typhoid bacilli, and appears to find a considerable difference in the behaviour of these two types. On this point, however, judgment must be cautiously withheld, for in the failure of so many other differential tests it is improbable that this one of agglutination by chemic reagents will prove specific. As to the significance of these experiments in explaining the *modus operandi* of the agglutination reaction no opinion can at present be formed, and this is equally true as regards their bearing upon the clinical side of Widal's test.—EDITORIAL, *Journ. Amer. Med. Assoc.*, September 18th, 1897.

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A NOTE FROM THE CLINIC

OF

SIR WILLIAM R. GOWERS, M.D., F.R.S.,

At the National Hospital for the Paralysed and Epileptic, Queen Square, London.

Habit Spasm.

GENTLEMEN,—I need not ask you to notice the spasmodic contortions of the boy before you; they are only too obtrusive. He is eleven years of age.

His history is that he was always an excitable child, and at seven years old he began to have attacks of shouting and screaming, clenching his hands in apparent fright, spitting and putting out his tongue. These symptoms continued, when they occurred, most of the day, and recurred for a year or so. They were varied by other strange aberrations. At one time he used to lick every object he could get hold of; he does not now seem to know why he did it, he can assign no motive for this strange manifestation of what may be called an animal action. He is said to be a "very good boy," whatever that may mean, but he has not been to school since he was seven years of age, though he can read. He is the youngest child, having one brother quite healthy. A step-brother is said to have had chorea.

Note the frequent shrugging of shoulders, the movements of the face and of the arms, and the

inspiratory guttural noise produced from time to time.

The case has been thought to be a peculiar example of chorea. This affection has nothing whatever to do with chorea; it is an extreme example of that which we may best call "habit-spasm." It is sometimes designated "habit-chorea," but, having no connection with chorea, it is best not to associate the two affections. It may be regarded as a crystallisation into a special form and the increase to a morbid degree of the restlessness of childhood, the restlessness which exists in the period of the first ten years of life. At this epoch, the motor structures having been fully developed, their functional stability is becoming established by use. The lower centres acquire structural completion and functional adequacy before the higher, by which they are controlled, and the relative difference in date of acquisition is manifested for a long time, by less control than is ultimately attained. Thus the condition exists for the development of these curious forms of morbid character and degree. If you watch any child under twelve years of age, you will see that there is no stillness for more than a few seconds at a time; sudden movement is continually occurring, and this restlessness is greater in children of neurotic tendency. This habit-spasm is sometimes met with in boys in peculiar persistence and severity. It is, however, generally the result of a special cause. In all such cases you should suspect the influence of masturbation. Boys with irritable nervous systems are, perhaps, more prone to acquire this habit, and on these its influence is greater. It increases the instability of the nervous system, and seems to cause the lower centres to be insubordinate in a special way. Remember that the habit sometimes begins very early, even at three or four years of age, but at any age, up to and at puberty, it may bring about the change in the state of the nervous system which underlies the severe forms of habit-spasm. My attention was first directed to that some twenty-five years ago by a remark which was made to me one day in private by Sir William Jenner, that the peculiar loud, barking, paroxysmal

sometimes met with in boys, and for which no ordinary treatment will do good, is always connected with masturbation. I have since found, repeatedly, the truth of the statement (as of most things I learned at that source of practical wisdom) that it is so, and I have learned to extend that statement to all respiratory disturbances in boys of that character and to most severe cases of spasm. So when this boy was admitted he was watched, and the suspicion of the presence of this cause was at once absolutely verified. We shall endeavour to arrest the habit, and you shall know the result.

[The symptoms ceased when this arrest had been secured by the customary operation.]

A Consideration of the Principles that should guide us in the Treatment of Abnormal Mechanical Conditions of the Hip-joint.

BY

W. ARBUTHNOT LANE, M.S.

GENTLEMEN,—I purpose considering with you to-day some points in the mechanics of the hip-joint and pelvis, in the hope that by the careful investigation of them we may be in a position to formulate some definite principles which should guide us in our treatment of disease or abnormality about the hip-joint, or in other words, of any deviation from the normal in the mechanical condition of the mode in which pressure is transmitted from the pelvis to the femur.

I think I will be able to show you at the present time that their treatment is founded upon our habits of imitation or mimicry of one another's surgical procedures, rather than upon any well-understood scientific mechanical basis.

We are inclined to copy more or less precisely the methods of others, occasionally going so far as to add to, or even to alter details. But we rarely study the principles which should govern surgical measures, and attempt to apply them for ourselves in our operative procedures.

We seem to dislike discussing mechanical principles, but are always ready to talk glibly and with action of our treatment and of our results.

The latter are frequently unreliable, sometimes because the surgeon deceives himself, and sometimes for other reasons.

Unfortunately for us, the descriptions of the physiology of the skeleton as contained in the text-books on anatomy are, as you are well aware, not only very scanty indeed, but what little there is, is usually utterly incorrect and misleading. Consequently the surgical practice that is founded on such an unsatisfactory basis is insecure and empirical rather than scientific.

If you examine the skeleton you see that the whole of the superjacent weight of the trunk is transmitted through the sacrum. In the active erect posture the lumbo-sacral joint is in a position of partial extension, while in the resting or easy erect position it is in one of considerable flexion.

In a paper* treating of the position of activity as illustrated by the anatomy of labourers, I showed that in habitual over-extension of the lumbo-sacral joint much of the superjacent weight is transmitted through the spinous process of the fifth lumbar vertebra to that of the sacrum. This is

Fig. 1 presents a condition the reverse of spondylolisthesis, namely, a displacement of the straightened or even anteriorly concave lumbar spine backward upon the sacrum, and the consequent transmission of a large part of the superjacent weight through enormously developed dense lumbar and sacral spinous processes. This condition results from carrying on the head, or the load being habitually borne in front of the chest or trunk.



evidenced by an excessive development of these processes, the formation of amphiarthroidal or arthrodial joints between them, and by the backward displacement of the body of the lumbar vertebra from that of the sacrum (see Fig. 1).

Such habitual over-extension acting as an evolutionary factor in my opinion brings about the dislocation of the first piece of the sacrum, as shown

* 'Guy's Hospital Reports,' 1885 and 1886.

in Fig. 2, which is described fully in the paper in the 'Guy's Hospital Reports.'

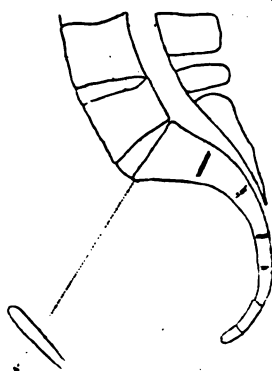


Fig. 2 represents a condition of the pelvis which at first sight closely simulates that associated with dorsal excurvation, namely, the diminution in the diameter of the conjugate of the brim of the pelvis, and the altered inclination of the facet on the first piece of the sacrum. This, however, is due to a separation or dissociation of the first piece of the sacrum to form one vertebra in excess above what are apparently the entire sacral series.

In the position of rest of the trunk, as represented by the skeleton of the inactive child and still more feeble old person, this joint is in a position of considerable flexion. This results in the displacement of the body of the fifth lumbar vertebra downwards and forwards off the sacrum, or in a yielding of the sacrum around a transverse axis causing a similar alteration in the position of this joint as regards the hip-joints.

When flexion of this articulation is assumed habitually by the labourer as an attitude of activity, a somewhat similar displacement of the last lumbar vertebra as regards the pelvic axes takes place, and all the superjacent weight is transmitted through the body, and none through the sacral spinous process, which with that of the fifth lumbar vertebra consequently is very ill-developed.



Fig. 3 represents the fourth and fifth lumbar vertebrae and the sacrum of the coal-heaver in vertical antero-posterior section, and illustrates the form of spondylolisthesis found in this class of labourer.

Figs. 3, 4, 5, 6, and 7 probably illustrate my meaning more clearly than the above description.

Remember, then, that in over-extension of this joint there is a tendency for a considerable propor-

tion of the superjacent weight to be transmitted through the spinous process, while in over-

Fig. 4 shows the form of spondylolisthesis that occurs in such labourers as carry heavy loads on their backs less heavy than those borne by the coal-heaver, and who pick them off and replace them on the ground. The arthrodial joint seen between the fifth lumbar vertebra and altered upper piece of the sacrum is produced by this greater freedom of movement.



Fig. 5 represents a vertical antero-posterior section through the spine in a position of activity, namely, the active erect posture; and Fig. 6 a similar section in a position of rest, which, when fixed by its habitual assumption, is called "dorsal excurvation of adult life" or "round shoulders."

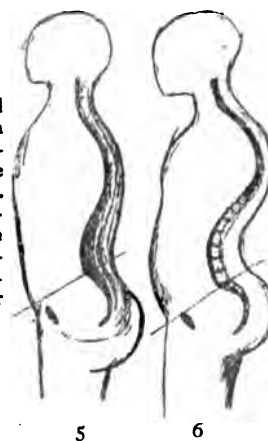
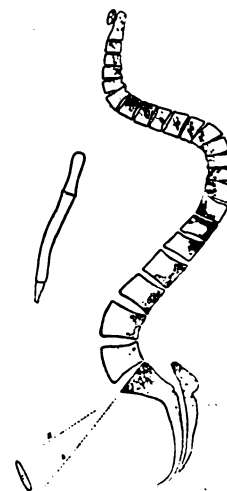


Fig. 7 shows the trunk of a feeble old subject in vertical antero-posterior section.



flexion very little, if any, is borne by this part of the sacrum.

There is another joint which is closely allied in its movements with that of the lumbo-sacral, namely, the sacro-iliac. These joints are flexed or extended simultaneously.

The sacro-iliac joint is of great interest, since the mechanical principle which is made use of in its anatomy is, as far as I know, applied in only one other place in the body, and there in a modified form.* While it is one of very great power, it readily undergoes changes from the normal type under the influence of abnormal pressure or variation in the habitual range of movement.

Being curious to find out what had been done recently by anatomists to render more clear its mechanics, which, in spite of the work of Matthews Duncan and others, seemed to me to be but little understood, I obtained the last English text-book on anatomy, namely, Morris's, and looked it up. In it I found the following:

"The movements.—It is quite clear from the nature of the osseous surfaces, from the wedge shape of the sacrum, and the manner in which it is locked in between the hip-bones, as well as from the amphiarthrodial character of the articulation, that *there can be no movement in the sacro-iliac joint.*

"While the joint serves the useful purpose of breaking shocks, the cartilage is too thin and too firmly fixed to the bones to allow even of appreciable yielding, such as occurs in the intervertebral discs. The double wedge shape of the sacrum, with its broader surface at the base and at the front, prevents dislocation from above downwards and from before backwards. The sinuous character of the opposing surfaces of the sacrum and ilium, the forward and inward direction of the fibres of the posterior sacro-iliac ligament which passes from the ilium to the sacrum, as well as the ilio-lumbar and lumbo-sacral ligaments, prevent forward displacement of the base of the sacrum; while the sacro-sciatic ligaments prevent the tilting backwards of its apex. *Thus rotation forwards is entirely prevented.* The anterior and downward displacements of the sacrum are prevented by the interosseous and posterior sacro-iliac ligaments, which pass from the ilia to the sacrum and suspend the latter, acting somewhat in the same way as the chains of a suspension bridge. They also bind the two bones more

tightly together, the greater the pressure the tighter the union. The suspension bridge arrangement of the sacro-iliac synchondrosis is admirably adapted to give strength to the pelvis."

Gentlemen, that represents the entire description of the physiology and mechanics of this joint as contained in this work under this heading. I presume you will be expected to understand, remember, and repeat it at the examination table.

I was pleased, however, to see that the editor does not consider the functions of the joints of such relative inferiority as to be put into very small print as is the case in 'Quain's Anatomy.'

Possibly, like myself, you may have failed to obtain a very clear grasp of the mechanical principles on which this interesting joint is constructed from the description I have just read to you from this anatomical text-book. To the ordinary observer it must appear very strange that such an elaborate and remarkable mechanical arrangement should have been evolved in order to *prevent the occurrence of any movement* between the sacrum and the iliac bones, when the same result could have been effected much more securely and simply by the fusion of these bones.

That the joint exists merely to serve the "useful purpose of breaking shocks," the writer himself while asserting seems to doubt, since he passes on to say that "the cartilage is too thin and too firmly fixed to allow even of appreciable yielding such as occurs in the intervertebral discs."

In the first place I will ask you to consider the mechanics of the sacro-iliac joint, since through it the whole of the superjacent weight of the body is transmitted. The principle involved in it consists in the apposition of concavo-convex surfaces retained in contact by means of the ligaments in front of and behind it, namely, the posterior sacro-iliac and the ligaments of the pubic symphysis. Roughly speaking, the articular surfaces are crescentic in form, the arc forming the concavity being part of a larger circle than that forming its convexity.

Force applied to the sacrum in such directions as to drive it directly downwards, or to cause it to rotate in a forward or backward direction around an axis passing through the attachment of the posterior sacro-iliac ligaments, brings about a separation of the corresponding articulating surfaces, since the prominence on one aspect emerges

* I refer to the calcaneo-cuboid articulation.

from the opposing concavity and travels along the inclined plane of an adjoining convexity. This movement of the sacrum upon the iliac bones is opposed and limited by the resistance offered by the ligaments already mentioned. The degree of movement permitted in a joint varies inversely with the age and vigour of the subject.

In the active erect symmetrical posture the sacrum is forced directly downwards in the vertical transverse plane in which the acetabular cavities lie. Also in the rotation of the sacrum which occurs in flexion and extension of the sacro-iliac joints, the pelvis is rotated around a transverse axis passing through both acetabular cavities till that portion of the sacrum which transmits the superjacent weight is brought into the same vertical transverse plane. Under these circumstances the three joints of the pelvis are practically fixed. If, however, the subject is standing in the active erect posture on one leg, the whole of the superjacent weight of the body is transmitted through one sacro-iliac joint to the iliac bone, and through it to the head of the femur, the pelvis having rotated around an antero-posterior axis in order to facilitate this transmission. This it does by bringing the sacro-iliac joint into a nearer vertical relationship with the hip-joint, while it also enables the other leg to be swung forwards free of the ground.

During the assumption of this posture the sacro-iliac joint on the same side is fixed, while the opposite sacro-iliac and the pubic are left comparatively loose. As a result of this, while the opposite leg is being carried forwards the innominate bone rotates around the transverse axis of pelvic rotation, its anterior superior spine descending to meet the advancing leg. This movement can naturally be best observed in feeble-bodied children with loose joints, in whom all joint movements are exaggerated. In cases in which the hip or lumbar joints are fixed, or are much limited in their range of movement, and in some varieties of labour—as, for instance, that from which Fig. 4 was obtained—the freedom with which the iliac bones move on the sacrum is very great, the sacro-iliac joints having acquired an arthrodial type.

The surgeon may with advantage recognise and make use of the possibility of increasing by practice the amount of movement in these joints in certain cases where he is unable to have recourse

to more radical measures. Additional evidence of this is afforded also by the fact that when one leg is shorter than its fellow, or is fixed at the hip-joint in a position of flexion, the sacro-iliac joint on the side of the shorter limb shows much more extensive pressure changes than that on the opposite side.

Do not think for one moment that the information that is supplied in the text-books represents the knowledge of the present day or even of the last generation, or that it will satisfy your examiners in obstetric medicine, or even at the higher anatomical examinations. The obstetric physician has known and has taught for years that considerable movement takes place in the sacro-iliac joint, and he makes use of it largely in his practice to the advantage of his patients.

I fancy the anatomist must have drawn his conclusions from a study of the skeletons of labourers, which form the large proportion of the subjects in our dissecting rooms, but one would imagine that even they would afford him more information. He certainly could not have studied the mechanics of this joint during youth, or under circumstances which show its variations to even greater advantage.

Many years ago Dr. Matthews Duncan described very fully this movement of the sacro-iliac joint as "nutration" of the sacrum. You will also find it figured in his most interesting works. If you look at Dr. Galabin's work on midwifery, a book with which you are well familiar with, you will find the following:

"In the adult woman, and especially in pregnancy, a synovial membrane exists between these surfaces, and a certain small degree of movement is permitted," &c.

In a paper * read before the Obstetrical Society I endeavoured to show that the evolution of these joints during pregnancy was brought about in a simple mechanical manner by the presence of a temporary load in front of the lumbar spine. This necessitates a considerable habitual extension of the lumbo-sacral joint, and a wide range in its limits of movement. The difference in the form of the sacrum in the two sexes is produced in this simple manner, as are also even more extensive variations in the thorax, which, probably

* "What are the Chief Factors that determine the Differences which exist in the Form of the Male and Female Pelvis?" 'Trans. Obstet. Soc.,' 1888.

for the reason that they are so very obvious, escaped the observation both of the anatomist and the physiologist.

The knowledge of this movement is occasionally of great importance. If you will take the trouble to look at a paper by Dr. Fothergill in the 'Medical Journal' of October 31st, 1896, you will find a description of what is called Walcher's position in parturition. Walcher found that he was able to obtain a variation of one centimetre, about one third of an inch, by bringing the pelvis from a position of extreme flexion to one of over-extension. This position of over-extension is obtained by placing the patient on the back and allowing the legs to hang freely down so that the feet do not touch the floor. By this means the innominate bones are fixed at the hip-joints by the over-extension of these articulations. This posture is now in use as a matter of routine in several German hospitals, where it is employed in all high forceps operations, in extractions after turning, and after perforation of the head,—all operations very materially assisted by any increase of the conjugate diameter of the brim of the pelvis.

Now what is the mechanical relationship of the pelvis to the femurs? In its movement upon the thighs the pelvis moves around two axes—a transverse one passes through the acetabular cavities, and around this axis the pelvis moves in opposite directions in the positions of extension and flexion. In the *symmetrical position of rest of the trunk* the pelvis rotates in such a direction as to over-extend the hip-joints, this movement being chiefly controlled and limited by the strong anterior ligament of the joint.

The iliac bones pass backwards, the ischial forwards.

Associated with this movement of the hip-joint the sacro-iliac, lumbo-sacral, and superjacent joints of the spine are flexed, the thorax being also retained in a symmetrical position of rest, or of expiration. This position, when fixed and exaggerated, is spoken of by the profession as "dorsal excurvation," and by the public as "round shoulders" (see Fig. 8).

As regards the pelvis, the superjacent weight of the trunk falls through the sacrum in a vertical transverse plane behind that occupied by the transverse axis of pelvic rotation, and so over-extends the hip-joint. It falls in front of the trans-

verse axis, around which the lower end of the femur rotates at the knee, over-extending this joint also.

This I have endeavoured to indicate diagrammatically. A B represents the pelvis, C the lumbo-sacral, D the sacro-iliac, E the combined lumbar joints and the line above the superjacent spinal column, A H the anterior ligament of the hip-joint,



Fig. 8 shows a typical example of the fixation of the symmetrical posture of rest of the trunk.

which resists downward displacement of the end of the lever at D by the superjacent weight of the trunk falling through it (Fig. 9).

In order that this position of rest be assumed normally, *it is necessary that there be a secure joint allowing of no vertical play in front of the vertical transverse plane along which the weight of the upper part of the body is transmitted through the sacrum, together with an arrangement to control the movement of the iliac bones backward upon it, representing the anterior ligament of the hip-joint.*

In passing from the easy erect to the active erect posture the pelvis rotates around the same axis, but in the opposite direction, until the transverse plane along which the superjacent weight of the trunk is transmitted through the sacrum corresponds to that occupied by the transverse axis of pelvic rotation. The sacro-iliac, lumbo-sacral and superjacent joints of the spinal column are ex-

tended, and the chest is in a position of inspiration. This is brought about by the action of muscles which take a very much larger share in its production than they do in the position of rest. In the latter posture the transmission of force is controlled chiefly by ligaments, and by structures performing, for the time being at least, functions of ligaments. This position of activity is recognised by the soldier as "standing at attention."

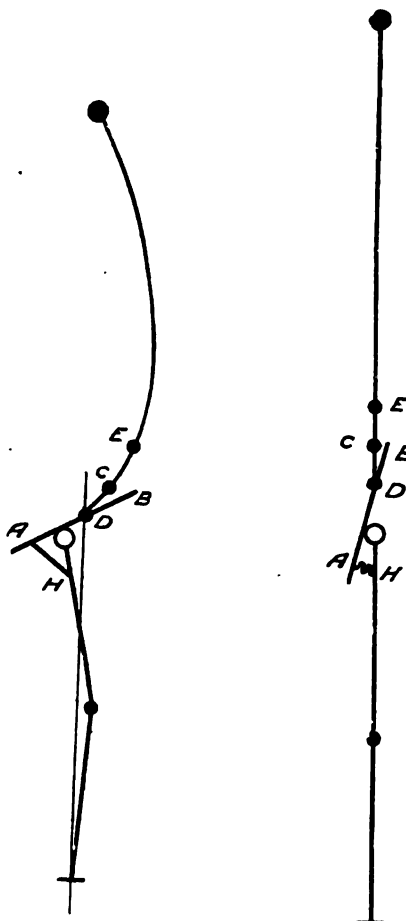


Fig. 9.

Fig. 10.

During locomotion in the erect posture this relationship is retained by a sequence of contractions of the muscles controlling the movements of the pelvis on the femur. That the positions of activity in the erect posture may be assumed satisfactorily it is necessary that it be possible to bring the point in the sacrum through which the superjacent weight of the trunk is transmitted into the same vertical transverse plane as that occupied by

the hip-joint, which must be of such a nature as to permit no vertical play of opposing bony surfaces, but shall, on the other hand, allow of the direct transmission of force from the innominate bone to the upper end of the femur. It is also requisite that this joint shall permit of rotation of the pelvis around the transverse axis passing through it.

In Fig. 10 this attitude is illustrated diagrammatically. The ligament of the hip-joint, A H, is relaxed, and the several articulations are in a position of considerable extension.

When the individual passes from the symmetrical position of rest of the trunk to the asymmetrical resting posture, in addition to this rotation of the pelvis around the transverse axis of pelvic rotation which was associated with the former, we have a rotation of it around an anterior posterior axis, the pelvis dropping on the side of the flexed limb. This rotation is controlled chiefly by the anterior ligaments of the hip-joint on which the body is supported, and by other structures, performing the function of ligaments, the weight of the trunk falling in a vertical antero-posterior plane internal to that occupied by the centre of the head of the femur through it is transmitted. The direction taken by the lever which is formed by the pelvis is therefore necessarily oblique, and not antero-posterior, as in the symmetrical position.

If a limb is congenitally shorter than its fellow,—a by no means uncommon condition in a slight degree,—deviation of the spine from an antero-posterior vertical plane is obviated by the shape of the last interarticular fibro-cartilage, which is wedge-shaped, the base of the wedge being directed to the shorter limb. If such a subject carry loads from an early period of life, this fibro-cartilage is rendered thin and flat like the others, but the last lumbar body assumes the wedge shape in obedience to the law that *the rate of bone formation at the several portions of an epiphysial line varies inversely with the pressure transmitted*. In this manner the spinal column does not deviate from the vertical antero-posterior plane in spite of the abnormal obliquity of the facet on which it is placed. If, however, the shortening be acquired, the pelvis rotates around the transverse and antero-posterior axes referred to in proportion to the difference in the length of the limbs.

Upon the oblique plane of the facet on the

sacrum the superjacent vertebræ of the vertical column form a curve at right angles to its surface. The joints of the lumbar spine and pelvis are over extended, and the condition called *lordosis* exists. By compensating for the shortening the movements of the pelvis and spine are restored to their normal character.

"Lordosis" is a term used by surgeons to comprise various conditions of over-extension of the sacro-iliac, lumbo-sacral and lumbar joints produced either by an excessive rotation of the pelvis around the normal or other transverse axis in such a direction as to effect a symmetrical over-extension of the above-mentioned articulations, or asymmetrically having associated with it a rotation of the pelvis around an antero-posterior axis. It is one of those unscientific, unmeaning terms wanting in definition and precision, like scoliosis, lateral curvature, kyphosis, flat-foot, and numbers of others which ought not to be used by a profession pretending to be scientific.

In the erect position the femur of the shorter limb occupies, as regards the pelvis, a position of flexion and abduction, the amount of rotation of the pelvis around the two axes varying directly with the differences in the length of the limbs.

This extension of the lumbar, lumbo-sacral, and sacro-iliac joints, with the abduction of the lumbar and lumbo-sacral, if one might so describe the lateral movements of these structures on each other present in acquired inequality of the length of the legs, may be lessened in great part by compensating for the shortening.

When the head of the bone or the upper end of the femur is ankylosed to the dorsum ilii above and behind the transverse axis of pelvic rotation, the degree of over-extension and abduction is still greater than in the last case, and the incapacity is increased correspondingly. In this it is not possible to lessen the curvature produced by over-extension of the joints by compensating for the shortening to anything like the extent that can be done when the head is fixed in the acetabulum.

Again, when the upper end of the femur moves upon the dorsum ilii in an arc limited only by the resistance offered by the fibres of the capsular ligament and the ligamentum teres, the pelvis rotates around its axes till the femoral attachment of the ligaments lie vertically along their insertion to the innominate bone.

When I was working at the changes in the skeleton that result in labourers from the pursuit of different occupations, or in other words the study of the fixation and exaggeration of various postures of activity, I examined several bodies in which fractures of the neck of the femur had been sustained, followed by non-union of the broken surfaces. I found two distinct varieties. The most common was that in which the stump of the neck was displaced on to the dorsum ilii above and behind the acetabulum, where it formed a joint, allowing an amount of mobility which was sometimes very considerable. In these bodies it was obvious that considerable physical depreciation had followed the receipt of the injury, and that there was much deformity consequent upon the alteration in the mechanics of the part.

It appeared to me that the dorsal displacement of the upper end of the femur had resulted from the misplaced activity of the surgeon, who by the principle involved in the use of the vertical foot-piece so rotated the femur inwards as necessarily to produce what was practically a dorsal dislocation. This I have explained fully to you in lectures on the treatment of fractures.

A few of these people appeared to have been sufficiently fortunate as to have escaped surgical treatment, or if they came under observation, were not subjected to the routine methods, and their legs were allowed to fall into a position of external rotation, so that the foot rested by its outer aspect on the bed. In these cases the stump of the neck formed with the ilium immediately beneath the anterior inferior spinous process a secure joint which allowed of no vertical movement of the bones on one another.

The condition of their bones and soft parts showed that the interference with the mechanics brought about by the formation of this new joint in front of and above the normal one was very much less than in the other case, and that the individual was able to continue some arduous and laborious pursuits in spite of it, more or less satisfactorily.

When the shortening was compensated for by the use of a thick sole, there was very slight alteration in the form of the lumbar spine and pelvic joints.

I have described these conditions briefly in a paper in the 'Medico-Chirurgical Transactions,' 1888, entitled "An undescribed method by which

the superjacent weight of the body is transmitted in fracture of the neck of the femur through an acquired ilio-femoral articulation, and the bearing of the principle involved on the surgery of the hip-joint."

Since this new joint is situated immediately in front of and slightly above the acetabular cavity, or the situation of the normal transverse axis of pelvic rotation, when the patient stands the pelvis rotates around an antero-posterior axis to an extent corresponding to the inequality in the length of its lateral supports, and there exists also such an amount of rotation around a transverse axis as is necessarily associated with the excessive rotation around an antero-posterior one.

This last movement brings the normal hip-joint and the new joint into the same vertical transverse plane, and as the latter allows of no vertical play of the bones on one another the superjacent weight is safely and securely transmitted to the femur of the shorter limb.

Compensation for shortening produces a rotation of the pelvis around an antero-posterior axis so as to render it symmetrical, or, more commonly, there is also a slight amount of rotation of the pelvis around a transverse axis in a direction the reverse of that which takes place after compensation for shortening is made with a joint on the *dorsum ilii*, and the lumbar, lumbo-sacral, and sacro-iliac joints are slightly flexed. The only discomfort from which such a patient suffers is the inability to move the thigh upon the pelvis with any great freedom; but this is a small trouble as compared to the mechanical disability of the patient who has a loose and insecure joint behind and above the acetabulum, such as exists in one variety of congenital dorsal dislocation of the hip-joint, in many cases of acquired dorsal dislocation, and in some forms of dorsal dislocation resulting from disease of the hip-joint.

Any attempt to compensate for the shortening in the latter deformity increases the amount of what is called "lordosis," which I have shown you is an over-extension of the lumbar, lumbo-sacral, and sacro-iliac joints, a position of great insecurity and mechanical disability. This occurs because the dorsal joint is situated behind the transverse axis of pelvic rotation, and in consequence the pelvis cannot be made to rotate satisfactorily around an antero-posterior axis.

Compensation for shortening throws an additional strain on a joint which is already insecure, and tends to increase its mobility in a vertical plane, together with the deformity and insecurity which result from it. This is well seen in that variety of congenital dislocation of the head of the femur upwards and backwards on the *dorsum ilii*.

I propose now to consider this congenital deformity first, and in doing so we should commence by asking ourselves these questions:

1. What is the anatomy of the condition?
2. What are its mechanics, or, in other words, its functions or physiology?
3. Can the head of the bone be restored to its normal position; and if not, on what principles should its treatment be based?

I do not propose to drag you through the literature of the subject, as I have already plodded through it and experienced how obscure, contradictory, and unreliable some of it is, but I will confine myself to submitting to you my own experience and arguments for your consideration and criticism. My operative experience is confined solely to the variety in which the head of the bone is displaced backwards, since I have seen but few instances of the forward displacement that called for active surgical interference, and these refused it. I have operated on several of the worst cases of the former that I have seen, sometimes on one side, sometimes on both sides.

1. *Anatomy*.—In nearly every one of them I found precisely the same structural conditions. The femur was, roughly speaking, normal in form, except that its neck was shorter than usual and its head was very decidedly flatter. It was just a condition as one would expect to result from the articulation of the head of the growing bone with a flat surface instead of with a uniform concavity, since the form of a bone varies with its function or physiology. There was a complete capsule attached both to the femur and to the innominate bone, whose areas of insertion corresponded more or less with the normal. The arrangement of the fibres was also very similar, since it was thickest above and in front, and thinnest below and behind; in other words, the structure and strength of its several parts varied directly with the strain sustained habitually by them. It differed from normal capsule chiefly in its greater length

thickness. The ligamentum teres presented similar conditions of elongation and hypertrophy.

On manipulating the leg, as during locomotion, it was clear that the capsule and ligamentum teres were the sole means by which the weight of the body was transmitted to the dislocated limb, and that the excessive strain thrown habitually upon these ligaments was responsible for their elongation and hypertrophy.

In but very few of these cases was there a normal or anything approaching a normal acetabulum, and no amount of cutting rendered it possible to replace the head of the femur in a cavity corresponding to the position of the acetabulum.

Very rarely an acetabular cavity was present, into which, after freely dividing the restraining structures, it was possible to replace the head of the femur, and to retain it there by means of wire sutures passing through the margin of the acetabulum and the capsular ligament. Even in the same subject the conditions may differ widely on the two sides. For instance, in an infant about two years of age there was present on the one side a well-formed articular cavity into which it was possible to place the head of the bone, and to retain it by sutures; while on the other, in the position of the acetabulum, there was merely a depression to whose level it was found impossible to bring down the upper end of the femur. As a general rule it would be true to state that the structures forming the hip-joint differ from the normal in direct proportion with the age at the time of operation. In other words, the older the child the more advanced are the changes which have been described above.

2. *Physiology.*—The capsule and ligamentum teres controlled the movement of the head of the femur upon the outer surface of the iliac bone in the form of an ellipse, the head being much nearer the iliac attachment of the ligaments when the body was in the recumbent posture, and most distant from it when force was directed upwards along the length of the shaft. This was apparently due to the fact that in the recumbent posture no strain was thrown upon the ligaments, which were relaxed, while the transmission of the superjacent weight of the trunk through the ilio-femoral ligaments separated their points of attachment to the utmost.

When this condition exists on both sides the pelvis ceases to exert any leverage action, since

there is no fixed point on which the head of the femur can act as a fulcrum, and consequently there is no such resistance to pelvic rotation as is exerted chiefly by the normal ilio-femoral ligament. That portion of the sacrum through which the superjacent weight of the trunk is transmitted has both in the active and easy erect posture to be brought into the same vertical transverse plane as that occupied by the heads of the thigh bones when supporting this weight. The degree of extension of the pelvic and lumbar joints must therefore be considerable. This condition is less marked where the dislocation only exists unilaterally, but is similar in its mechanics on the affected side.

On exerting traction on the femur in a direction away from the joint, the capsule offered no resistance; but movement in this direction was opposed by all the muscles attached to the femur, by the fasciæ and other soft parts.

In order to remedy this very faulty mechanical condition it was clear that the best method would be to replace the head of the bone, or what represented the upper articulating surface of the femur, in a cavity occupying the position of the normal acetabulum, or, in other words, in the transverse axis around which the normal pelvis rotates. Then by compensating for the shortening the pelvis could be rendered symmetrical.

I would for one moment call your attention to the form of the upper end of the femur in this condition, since some writers seem to speak of it as if it were normal in form when they discuss the possibility of replacing it in an acetabulum which is presumably also of the natural shape. This may be true in certain cases; but it neither corresponds with my experience of the conditions found in the vast majority of patients operated on, nor with those which arguments by analogy afford.

As far as I know, a growing bone remains normal only so long as it performs its normal functions. Alter the mode in which pressure is transmitted through it and you change its form in a definite manner, and in one in which you, aware of the physiology of the skeleton, could readily preconceive by a study of the alteration in the pressure from the normal, without any experience of the anatomy of the deformity.

In some of the cases the surface covered with cartilage and synovial membrane does not cor-

respond to the position of the normal head of the bone, and much of what ought to correspond to the head has no such covering. I found that however freely I divided the soft parts—and that I did not hesitate to do as extensively as possible without destroying the vitality of the limb,—I was unable to place the upper end of the femur, which was trimmed down to resemble and perform the function of the normal head, in a cavity cut in the position of the acetabulum.

Thereupon, remembering the great mechanical advantages possessed by a secure joint beneath the anterior inferior spinous process of the ilium in front of the transverse axis of pelvic rotation over a loose one behind and above it on the dorsum illi, I determined to evolve such a joint in these cases, which to my great satisfaction I did with a most happy result.

The method of operating is the following: An incision is made backwards from the anterior inferior spinous process along the upper limit of the femur. The structures superficial to the capsular ligament are cut through, and the front of the ilio-femoral ligament freely exposed and its limits and attachments clearly defined. This ligament is then divided at its attachment to the innominate bone, being left as a long strong plane in connection with the femur. The *ligamentum teres* is cut away from the head of the femur.

The soft parts are cut through till it is possible to rotate the articular surface of the femur outwards and bring it forwards to the same horizontal plane as the anterior inferior spine. The upper end is so shaped with a chisel and file as to form a blunt cone.

A cavity is cut by means of a gouge in and beneath the anterior inferior spinous process, and the newly constructed head is placed in it. It is retained there by sewing the anterior ligament to the bone and the rectus tendon and to the soft parts in the vicinity, silk being usually used for the purpose. This is done very thoroughly and carefully, so that the fibres of the ligament will be directed to the best advantage. If the head cannot be securely retained in this manner, as is occasionally the case, a thick silver wire is passed through the trochanter, neck and head of the femur, and through the floor of the concavity cut in the ilium, and so fixed as to allow of free movement of the bones on one another around the wire as an axis. The wire

is removed subsequently, after the joint has reached a satisfactory stage in its evolution. But, as I have already stated, wire is rarely required, for the reason that the anterior ligament affords in the large majority of cases a means by which the head can be retained securely in position.

You will readily recognise the importance of performing every detail of this operation carefully, and of attending to it till a new ball-and-socket joint has been developed by systematic movements

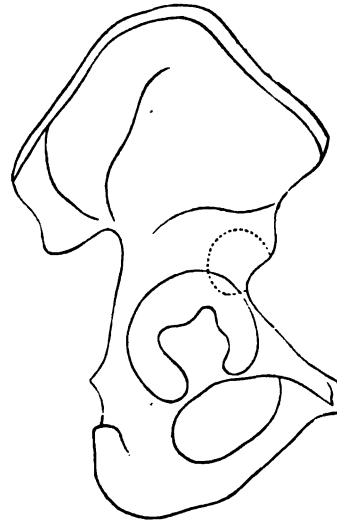


Fig. 11 shows the position of the cavity which is cut in the innominate bone.

extending over a variable but considerable period. Any failure to obtain a good moveable joint is due to insufficient movement after the operation, providing that it has been properly performed.

In choosing the time to operate I would be guided by the knowledge of the following law, which I think is true. *The younger the subject the sooner is a new joint developed, and the more perfect are the structure and mechanism of the articulation evolved.*

I cannot impress the importance of this too much upon you. Unfortunately there is a mistaken idea that young children bear operations badly. Nothing can be more untrue. This cannot be better illustrated than by the operation of cleft palate, to which I called your attention recently. The only obstacle that early life affords is the comparative difficulty of securing cleanliness. Remember, then, that *if you want to obtain*



Fig. 12 shows the thigh flexed and abducted.



Fig. 14 shows the trunk supported securely on the new joint, the pelvis being retained in a symmetrical position.



Fig. 13 shows the thigh flexed.



Fig. 15 shows the amount of adduction of which the hip-joint is capable in the erect posture.

moveable joint in any condition whatever, the earlier you operate the better will be the result.

In cases of acquired dorsal dislocation of the hip in childhood the mechanical conditions are similar, though more marked, owing to the greater length of the head and neck. This produces a considerable amount of flexion of the thigh on the pelvis, with a correspondingly greater amount of over-extension of the pelvic and lumbar joints. The same factor causes a degree of rotation of the femur inwards which is unknown in the congenital variety. In these cases, if a sufficient time has elapsed to render it impossible to bring the head of the femur down to the position of the acetabulum, a new joint must be formed beneath the anterior inferior spine. In cases of some standing such an operation requires more extensive division of parts than in the congenital variety.

Figs. 12, 13, 14, and 15 represent the first case of this sort on whom I operated. The photographs were taken in September, 1892, two years and five months after the operation. The new joint was then perfectly secure, having undergone no displacement and permitting all the movements present in the normal hip-joint. The boy was nine years old when operated on, and the dislocation had been produced three years before. He has improved still further during the last five years, and he can now run about as well, and his father says as fast, as any other boy of his age, and to all appearances the new joint is mechanically perfect.

The next condition I would call your attention to is that of dorsal dislocation resulting from disease of the hip-joint, usually tubercular. In this group we have to deal with a femur flexed and adducted upon the pelvis, and allowing either of limited movement of the bones on one another, or of practically no movement whatever. You are, unfortunately, very familiar with these cases, and are well aware of the great mechanical depreciation that results from this deformity.

These cases I treat on precisely the same principle as the last. You must remember that as there is very rarely any useful anterior ligament present, the head of the bone must be retained in position by means of wire till a new joint has been evolved, when the wire can be removed. Also that there is much more destruction of the head and the neck of the femur, and latent organisms may be exposed. Any tubercular

material should be carefully looked for and eradicated, and the child should be put upon some germicidal treatment. I use for this purpose mercury, arsenic, and the iodides of ammonium, sodium and potassium, the dose of arsenic being steadily increased. On this subject I would refer you to an earlier lecture on tubercular disease of joints in the 'Clinical Journal,' July 4th, 1894.

The procedure is indicated by the following case. L. H—, æt. 9 years, was operated on on May 6th, 1897. Three and a half years before he fell out of a van, and has been lame and in pain ever since. The left leg was two inches shorter than its fellow, the upper end of the femur was dislocated upwards and backwards on the dorsum ilii, the thigh was flexed and adducted, and movement was very limited and painful.

Extensive disease was found, and a sequestrum was removed. The neck of the femur was freed from disease, and was suitably trimmed. It was then secured by wire passing through it, and a cavity cut in and beneath the anterior inferior spinous process. The acetabular cavity, or rather the place corresponding to it, was rendered as free from the tubercular material as possible. A plug of iodoform gauze was inserted.

The photographs were taken on July 6th, two months after the operation. Fig. 16 represents the boy holding the thigh flexed. The long exposure has resulted in some blurring of the outline, the boy having moved slightly. Fig. 17 indicates the amount of passive flexion of which the joint was capable. The photographs also show that the adduction of the joint, which originally increased the deformity very considerably, has been completely removed.

Figs. 18 and 19 illustrate the condition of a child, A. W—, æt. 4½ years, operated on on February 19th, 1897, in which the disease producing the deformity had been cured for a considerable time.

When five weeks old he had an acute inflammation of the hip-joint, which resulted in the bursting of a large abscess in the upper limit of the inner aspect of the thigh, where the cicatrix can be seen in the photograph, and in the dislocation of the upper end of the femur upwards and backwards on to the dorsum ilii, where the trochanter was within a couple of fingers' breadth from the crest. The child before the operation was only able to

get about in the most clumsy way imaginable, the leg being three and a quarter inches shorter than



Fig. 16.



Fig. 17.

its fellow, and a mere flail. Walking exercise was quite out of the question.

The operative procedure was that already

described. The articular surface was flat, and rested upon a short stumpy neck. The acetabular cavity was obliterated, a slight dip in the bone indicating its original position. There being but a very imperfect capsular ligament, besides utilising



Fig. 18.

it it was necessary to pin the head in position with thick silver wire, which was removed at the end of four weeks, when the joint appeared sufficiently secure to allow of its removal. It was possible in this case to commence movements very early, as there was no suspicion of the presence of latent organisms.

These photographs were taken on the 27th March, about six weeks after the operation, and by that

time a thoroughly efficient joint allowing of very free movement had been developed, and about two inches and a half of the shortening had been removed. With the small amount of shortening compensated for, the child could stand securely and gracefully, and could walk very fairly indeed,

The following is abstracted from the report of the boy's condition on August 9th, 1897:

"The patient can walk and run well except for some tilting of the pelvis towards the shorter limb. This can readily be removed, since the left leg is now only three quarters of an inch shorter than its



Fig. 19.

considering that he has practically only just learnt to do so. This patient has been converted from a condition of complete physical inability into a state of what will soon be almost complete physical efficiency by the application of the mechanical principles enunciated in this lecture.

fellows. Flexion is perfect, as he can kiss his left knee with ease. The thigh can be abducted to an angle of 60° . Both active and passive rotation are as free as on the right side. The new joint is perfectly secure, and allows of no vertical play."

Considering the fact that before the operation

the child dragged the leg, and was unable to use it with any security for purposes of locomotion, and that only five months have elapsed since the operation, I do not think one could imagine a better result.

In the last case the femur moved freely on the dorsum ilii. In cases of fibrous or bony ankylosis it is necessary to cut through the attachment with chisels, leaving as long a neck to the femur as possible, and shaping its extremity so that it will perform the functions of a head.

When the hip-joint is ankylosed either in an extended position, or, as is very much more usual, in a position of considerable flexion and adduction, the physical disability is very great. In such, after cutting the upper end of the femur out of the acetabular cavity and shaping it, it should be securely fixed in a cavity corresponding to the upper and anterior portion of the original acetabulum and the bone above it, or in one cut beneath the anterior inferior spinous process.

The former is possible in such cases of ankylosis as are associated with a femur occupying a position of extension on the pelvis, but when the relationship is one of flexion and adduction the shortening of the soft parts renders it impossible to bring the head down to the limits of the acetabulum. To develop a joint in these cases often makes a considerable demand on the patience, skill, and perseverance of the surgeon; and if much care be not bestowed on the careful carrying out of the details of the operation, and of the subsequent treatment, the result may be by no means satisfactory.

I have under observation at the present time several cases of dorsal dislocation from disease of the hip-joint, in which the patients have been restored to an active life with a good working ilio-femoral articulation, having been originally totally incapacitated and dreadfully deformed by the fixation of one or both limbs in a position of considerable flexion and adduction.

I know no operation in surgery that has afforded me more satisfaction or the patient more advantage than this procedure.

If you once grasp the mechanical principle on which these methods of operating are based, their application will ensure you a moveable and efficient limb in the young subject, providing you devote much time and attention to the evolution of the new joint.

I do not propose in this present lecture to consider the operative treatment of hip-joint disease other than to point out to you that it must be founded on the same clearly defined mechanical basis. It seems to me only the other

day that surgeons performed an operation which they were pleased to call excision of the hip-joint. It consisted in cutting off the upper end of the femur by dividing it transversely at the greater trochanter. The broom-

the shaft was left unattached and to form for itself some sort of head somewhere, with a result that was disastrous, and from an æsthetic point of view disgusting and appalling. More recently surgeons have confined themselves to cutting through the neck of the bone, removing the head and part of the neck. They prided themselves much on the supposed advantages they gained in doing this through an anterior incision, and in obtaining what was called primary union. This childish fancy was marred mechanically by the rotating the shaft of the femur from the position of rest through an angle of forty-five degrees or more, till the inner margin of the foot occupied a vertical position. This being done after the wound was closed, the surgeon failed to observe that if he did not at once produce a displacement of the stump of the neck of the bone on to the dorsum ilii, it was placed in such a position that it occurred very shortly.

A recognition of the fallacy of the vertical foot-piece, the replacing such a set operation by the careful removal of diseased parts, and the use of iodoform* as a plug for cavities which used to be excised, has improved very considerably the physical efficiency of these cases after operation. In deciding in acute cases of hip-joint disease, which on the one hand should be treated by careful erosion and plugging, the head or stump of the neck being replaced in the acetabular cavity, and which on the other hand can be most effectively met by the formation of a new joint above and in front of the acetabulum beneath the anterior inferior spinous process, you must be guided largely by the extent of disease present, the presence or absence of dislocation, the age of the patient, and the degree of irremovable flexion and adduction of the joint.

It is obviously better to secure at once a moveable joint, in a good working position, with a small amount of shortening that can be readily compensated for, than to obtain a limb even of full length in any position of flexion or extension allowing of no movement at the hip-joint.

Do not lose sight of general principles, and do not let details of such trifling importance as the position and direction of the incision, the particular structures cut through, or the rate of closure of the wound, occupy your mental vision to the exclusion of the former, which are vastly more important, and to which these, though deserving of thoughtful attention, bear but a very subsidiary relationship.

* One of the best applications of iodoform in surgery ('Lancet,' July 15, 1893).

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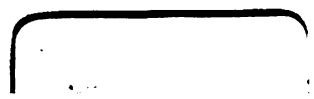
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